

# Geriatrics and the bladder: From a neurourological point of view

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## Abstract

**Background:** Bladder problems in older individuals are very common; however, their pathophysiology and management remain poorly understood. **Objective:** This article examines geriatrics and the bladder from a neurourological perspective. Aging notably impacts the brain. Among octogenarians (over 80 years old), the following estimated prevalences are reported: white matter disease (WMD) at 80%, Alzheimer's disease (AD) at 33%, and dementia with Lewy bodies (DLB) at 8%. Both isolated and combined pathologies are frequent, with AD+WMD being the most common combination. WMD is considered the primary pathological cause of overactive bladder in the elderly, while AD and DLB also contribute, though to a lesser extent. In advanced dementia cases due to AD or DLB, functional urinary incontinence may occur because of immobility, cognitive impairment, and loss of initiative. Early overactive bladder may be managed with  $\beta_3$  adrenergic receptor agonists and anticholinergics with minimal blood–brain barrier penetration. Advanced functional urinary incontinence may be addressed with behavioral strategies (e.g., prompted/timed voiding), toileting/environmental optimization, gait rehabilitation, and therapies targeting cognition and mobility. These conditions are potentially treatable in geriatric patients. **Conclusion:** This review highlights the unique integration between aging brain pathology and urodynamic findings, as well as functional urinary incontinence.

**Keyword:** Geriatrics, Overactive bladder, Functional incontinence, White matter disease, Alzheimer's disease, Dementia with Lewy bodies

## 1. Introduction

The proportion of older individuals (aged >65 years) increased worldwide from 5% in 1974 to a projected 10.3% by 2040,<sup>1</sup> necessitating increased medical care, particularly for age-related disorders.<sup>2</sup> Geriatric syndromes include dementia, delirium, behavioral disorders, gait difficulty, falls,<sup>3</sup> aspiration, and autonomic dysfunction.<sup>2</sup> Among these syndromes, the brain is known to contribute significantly to dementia and gait difficulty. While major autonomic disorders, such as bladder dysfunction, may have multiple etiologies, neurologic factors are increasingly recognized as underlying contributors.<sup>4</sup> This review summarizes (i) overactive bladder (OAB) and functional urinary incontinence (fUI) from the viewpoint of brain pathologies in older individuals, and (ii) the brain mechanisms and management of OAB and fUI.<sup>5</sup>

## 2. Overactive bladder and functional urinary incontinence: From the viewpoint of brain pathologies in older adults

In this section, we provide an overview of OAB and fUI from the viewpoint of brain pathologies in older individuals.

Recent surveys indicate that common brain diseases in older individuals include white matter disease (WMD), Alzheimer's disease (AD), and dementia with Lewy bodies (DLB), which is associated with Parkinson's disease (PD). Among these, WMD is the most prevalent, with an estimated frequency of 80–95% in octogenarians (>80 years old) and 58.3% in individuals aged 40–90.<sup>6,7</sup> In contrast, AD is the most common neurodegenerative disease, followed by DLB and

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other conditions. The reported ratio of AD to DLB (including PD with dementia) varies across countries: 14.6:1 in Japan (multicenter study<sup>8</sup>), 11.2:1 in the United Kingdom (nationwide study<sup>9</sup>), and 8.1:1 in the United States (nationwide study<sup>10</sup>), clinically; and 60.6%:24%, 55.5%:21.7%, and 52%:18%, respectively, pathologically, based on the Adult Changes in Thought Study,<sup>11</sup> National Alzheimer's Coordinating Center Study,<sup>12</sup> and a Japanese study.<sup>13</sup>

In our previous neuroimaging-assisted studies, the ratio of WMD, AD, and DLB/PD in octogenarians was approximately 80%, 33%, and 8%, respectively<sup>14</sup> (Figure 1). Furthermore, recent clinical and pathology studies indicate that both isolated and combined brain pathologies are common in older individuals. Among these, a combination of AD and WMD is the most prevalent,<sup>14</sup> but other combinations of WMD, AD, and DLB/PD are also observed.<sup>14</sup>

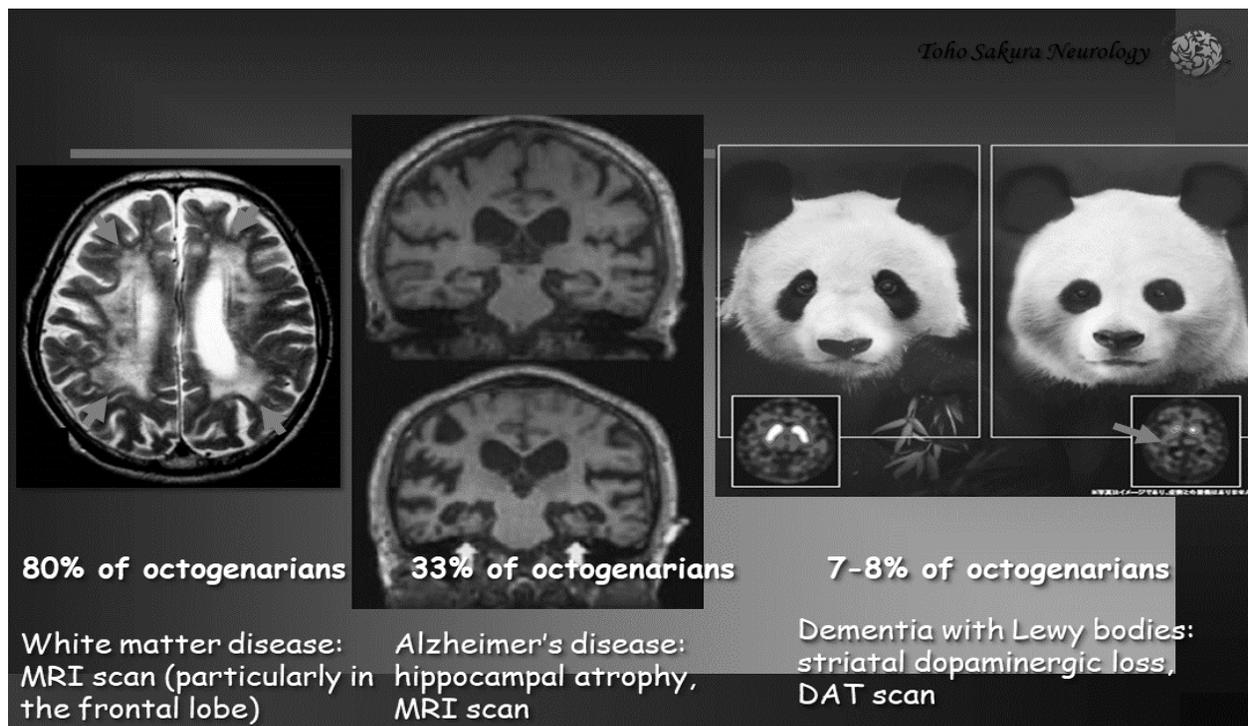
## 2.1. White matter disease, the major cause of overactive bladder, is also associated with functional urinary incontinence due to gait difficulty

White matter disease, also known as cerebral small-vessel disease or microvascular ischemic disease, is the most common brain disorder. In individuals with WMD, brain magnetic resonance imaging (MRI) typically shows bilateral symmetric hyperintense white-matter lesions. The extent and severity of these MRI-detected lesions can be graded on a

0–4 scale (Figure 2). Grade 0 is assigned when no lesions are detected. Grade 1 indicates punctate high-signal foci in the white matter located just above the frontal horns of the lateral ventricles. Grade 2 is used when lesions appear elsewhere but remain limited to the immediate subependymal region of the ventricles. Grade 3 denotes both periventricular lesions and separate, discrete deep white-matter foci of abnormal signal. Grade 4 is reserved for cases in which these discrete foci have enlarged and merged.

White matter disease results from systemic atherosclerosis and is therefore closely linked to atherosclerotic risk factors, including the cardio-ankle vascular stiffness index, hypertension, dyslipidemia, diabetes, smoking, alcoholism, other atherosclerotic organ diseases (e.g., myocardial infarction, ischemic cardiomyopathy, peripheral artery disease/arteriosclerosis obliterans/foot amputation, and chronic kidney disease/hemodialysis<sup>15</sup>), and an increased risk of acute stroke. Accordingly, management of WMD aligns with standard atherosclerosis risk-factor management, including lifestyle modifications, such as avoiding overeating and smoking. Because patients with WMD often have comorbid cerebral microbleeds—commonly due to atherosclerosis and usually clinically silent, occurring in approximately 50%—clinicians should exercise caution when initiating antiplatelet therapy in these individuals.

Cardinal features of WMD include OAB and gait



**Figure 1.** Three common brain diseases in older adults. The image is original by the author. Abbreviations: DAT: Dopamine transporter; MRI: Magnetic resonance imaging.

disturbance (vascular parkinsonism; pure lower-body akinesia; slow, short-stepped gait,<sup>16,17</sup> sometimes with an ataxic component). In contrast, dementia severity in WMD (vascular dementia) is generally mild, with Mini-Mental State Examination (MMSE) scores typically >15–18/30 (normal >24/30).<sup>18,19</sup> Our research group has shown that in individuals with WMD, symptoms emerge and progress in the following order: first OAB, then gait impairment, and subsequently cognitive decline—changes that are particularly notable in those with WMD grade >2 on the 0–4 MRI scale.<sup>20</sup>

Although cortical WMD appears diffuse on MRI, detailed pathological studies have demonstrated that the frontal lobe is the most severely affected region, showing marked hypoxic–ischemic injury to oligodendrocytes.<sup>21</sup> This finding aligns with evidence from MRI volumetry, which reveals prominent frontal lobe atrophy, while glucose metabolism,<sup>22</sup> N-acetyl-aspartate levels,<sup>23</sup> and brain acetylcholine concentrations<sup>24</sup> are most reduced in this region. Consistently, brain perfusion studies show the greatest reduction in the frontal lobes of individuals with WMD.<sup>25,26</sup> These observations likely explain why OAB, gait disturbance, and executive dysfunction—typically in the absence of severe dementia—are characteristic features of WMD.

## 2.2. Alzheimer's disease, the major cause of functional urinary incontinence due to dementia

Alzheimer's disease is the leading cause of dementia in older adults.<sup>27</sup> The pathological hallmarks of AD are neurofibrillary tangles composed of phosphorylated tau and extracellular amyloid plaques. In recent years, tau, amyloid, and other biomarkers have become measurable through cerebrospinal fluid analysis, positron emission tomography, and even peripheral blood assays.

In individuals with AD, brain MRI typically shows parahippocampal atrophy, while brain single-photon emission computed tomography (SPECT) perfusion imaging demonstrates reduced blood flow in the hippocampus, parietal cortex, and precuneus.<sup>28</sup> The characteristic clinical feature of AD is the amnesic syndrome of the hippocampal type (memory impairment), although variants such as posterior cortical atrophy and logopenic variant primary progressive aphasia may also occur. These symptoms often impair independence in activities of daily living. In advanced AD, the MMSE score may decline to 0/30, with patients becoming completely dependent. Behavioral and psychological symptoms—including delusions, hallucinations, irritability, and insomnia—are also common.

Differential diagnosis requires ruling out medical causes of cognitive decline, such as hypothyroidism. Delirium (systemic inflammatory encephalopathy triggered by surgery and

aspiration) may occur on top of dementia. Treatment options for AD include lecanemab and donanemab—IgG1 antibodies designed to clear brain amyloid and now approved in the United States and globally. Widely used cognitive enhancers, such as central acetylcholinesterase inhibitors and memantine, assist with symptom management and functional preservation. Management of behavioral and psychological symptoms of dementia requires targeted interventions.

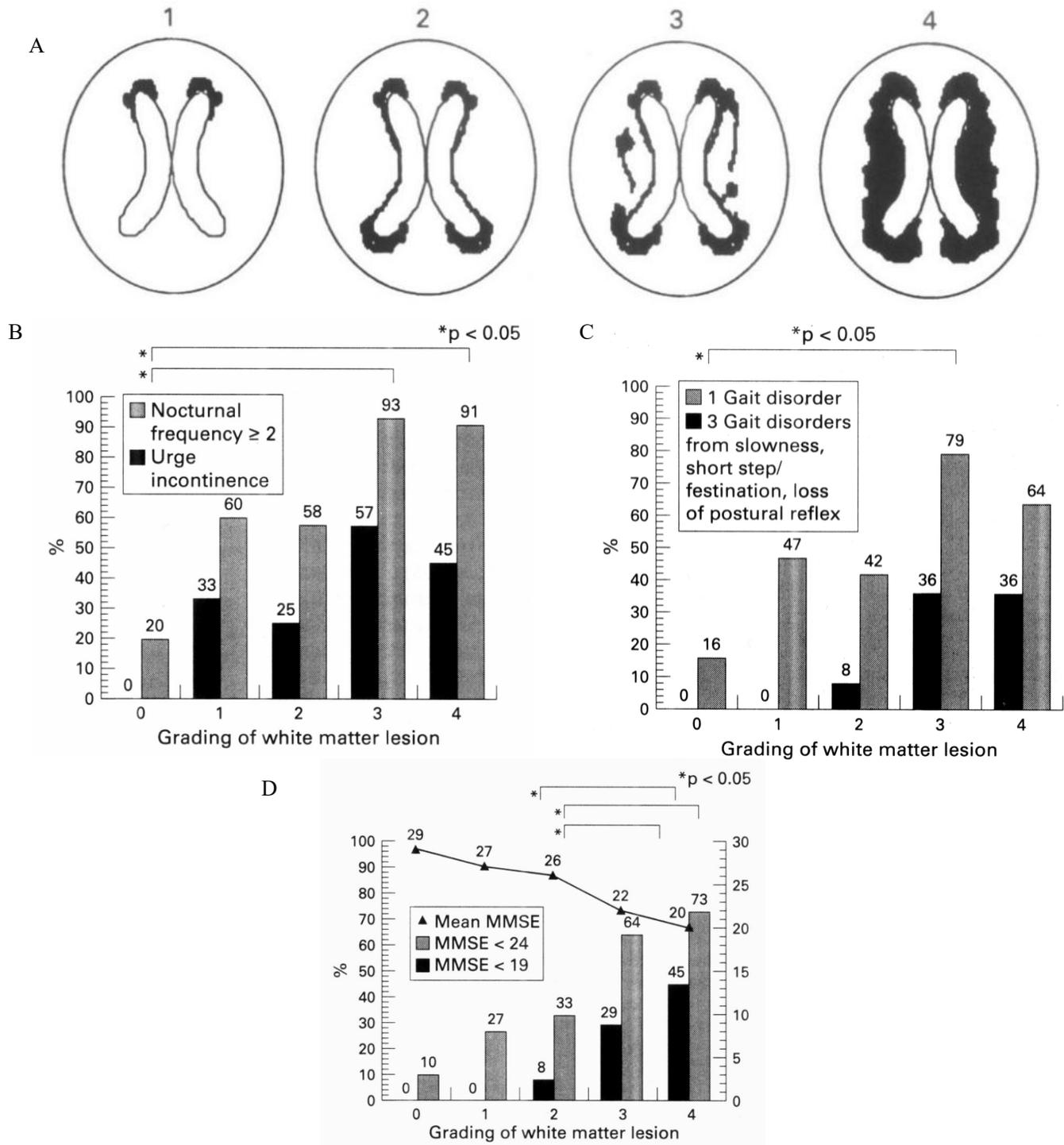
Alzheimer's disease also occurs as young-onset AD (age 40–50 years), which is regarded as a pure form of the disorder. Unlike cases with severe dementia in older adults, young-onset AD typically presents with minimal gait impairment or OAB, likely due to the absence of comorbid WMD commonly observed in geriatric populations. To date, no urodynamic studies have been conducted in this younger group.

In contrast, when AD is carefully distinguished from WMD in older individuals, pure AD shows OAB and urodynamic detrusor overactivity in up to 40% of cases—lower than the approximately 80% OAB prevalence reported in WMD. This difference likely reflects the less frequent involvement of the frontal cortex in AD.<sup>29</sup> In such cases, loss of central acetylcholine contributes not only to cognitive decline but also to OAB, because central acetylcholine exerts a bladder-inhibitory effect, whereas peripheral acetylcholine has a bladder-facilitatory effect.<sup>29</sup>

## 2.3. Dementia with Lewy bodies: Contributions to overactive bladder, functional urinary incontinence, and gait disturbance

Dementia with Lewy bodies is the second most common degenerative cause of dementia. Because DLB and PD share the same  $\alpha$ -synuclein–positive Lewy body pathology,<sup>30,31</sup> the term “Lewy body disease” is often used to encompass both conditions.<sup>32</sup>  $\alpha$ -Synuclein and other biomarkers can now be measured through cerebrospinal fluid analysis, positron emission tomography, and peripheral blood assays. DLB/PD generally does not show distinctive abnormalities on conventional brain MRI. However, <sup>123</sup>I-ioflupane (FP-CIT) dopamine transporter (DAT) SPECT and <sup>123</sup>I-metaiodobenzylguanidine (MIBG) myocardial scintigraphy can detect characteristic changes. DAT imaging reflects dopaminergic loss in the striato-nigral pathways, while MIBG scintigraphy indicates cardiac noradrenergic depletion. Additionally, DLB/PD are major contributors to gait disturbance.

Recent studies have shown that DLB may also present with a lower-body, pure-akinesia motor phenotype resembling WMD.<sup>33</sup> Individuals with DLB develop dementia, with the most advanced cases showing MMSE scores of 0/30. Unlike AD, cognitive impairment in DLB fluctuates and is



**Figure 2.** Cerebral white-matter lesions and overactive bladder. (A) Schematic presentation of the grading of white-matter lesions on MRI (according to Brant-Zawadzki *et al*, AJNR 1985; 6: 675-682). Grade 1: punctate foci of high signal intensity in the white matter immediately above the frontal horns of the lateral ventricles. Grade 2: white-matter lesions present elsewhere but confined to the immediate subependymal region of the ventricles. Grade 3: periventricular lesions together with separate, discrete deep white-matter foci of abnormal signal. Grade 4: discrete white-matter foci enlarged and became confluent. (B–D) Percentage of (B) urinary dysfunction, (C) cognitive impairment, and (D) gait disturbance according to the disorder severity and white-matter lesion grade on MRI. Reprinted with permission from Hanyu *et al.*<sup>26</sup> Copyright © 1999, BMJ Publishing Group Ltd. Abbreviations: MMSE: Mini-Mental State Examination; MRI: Magnetic resonance imaging.

frequently accompanied by visual hallucinations. In addition, DLB/PD often involves rapid eye movement sleep behavior disorder—manifesting as sleepwalking or dream enactment—due to impaired muscle atonia during rapid eye movement sleep. Severe constipation is also common in DLB/PD and may lead to intestinal pseudo-obstruction, paralytic ileus, volvulus, or intussusception requiring emergency surgery.<sup>34</sup> Treatment options include levodopa, dopamine agonists, zonisamide, istradefylline, and device-assisted interventions such as deep brain stimulation of the subthalamic nucleus. Levodopa may worsen hallucinations when used to treat gait problems. Psychotropic medications may be required for behavioral and psychological symptoms of dementia, but they can exacerbate motor symptoms, making careful medication balancing essential.

Overactive bladder occurs in approximately 60% of PD cases and 70–80% of DLB cases, with symptoms generally more severe in DLB. In DLB, OAB is more frequently accompanied by urinary incontinence due to reduced bladder capacity and detrusor overactivity. This is because the brain's nigrostriatal pathways regulate not only motor function but also bladder function via the D1 dopaminergic (bladder-inhibitory) pathway. OAB and detrusor overactivity closely correlate with abnormalities seen on DAT scans.<sup>35</sup> Additionally, severe dementia in DLB can lead to fUI, where patients are unable to reach the toilet due to spatial disorientation or loss of initiative, resulting in total dependence, similar to that observed in AD.

## 2.4. Other brain diseases in older individuals

Other brain diseases in older individuals that potentially contribute to bladder dysfunction are listed below.

### 2.4.1. Normal pressure hydrocephalus: Clinical signs mimic those of white matter disease, including an overactive bladder

Normal pressure hydrocephalus (NPH) is a less common but potentially treatable cause of OAB, urinary incontinence, gait difficulty, falls, and mild dementia (executive dysfunction).<sup>36</sup> While the exact cause of NPH remains uncertain, an increase in leucine-rich glycoprotein in the cerebrospinal fluid has been documented. MRI scans reveal the presence of disproportionately dilated cerebral ventricles, tight high convexity with dilated Sylvian fissures, and a narrow callosal angle. A positive spinal tap test may predict successful outcomes of shunt surgery. Cerebral blood flow is markedly decreased in the frontal cortex, similar to that seen in WMD; therefore, the clinical features of NPH can mimic WMD. The incidence of WMD versus NPH is approximately 10:1. Hence, potentially treatable NPH should not be overlooked in patients clinically suspected of having WMD, and brain imaging should be performed. Like WMD, OAB and gait

difficulty often precede dementia, and OAB precedes urinary incontinence in NPH.

Overactive bladder is found in up to 90% of NPH. OAB and its recovery in NPH strongly correlate with frontal hypoperfusion, suggesting that the frontal cortex contributes significantly to the occurrence of OAB in this disorder.

### 2.4.2. Alcoholism: A cause of overactive bladder

Alcoholism or alcohol abuse is a relatively uncommon cause of dementia, affecting 2–5% of octogenarians. The acute form is known as Wernicke encephalopathy, while the chronic form is referred to as alcohol-related dementia. OAB is a frequent feature in this condition; however, alcoholism can also mimic multiple system atrophy by causing postural hypotension, urinary retention, and constipation due to vitamin B1 deficiency-related peripheral neuropathy. Clinicians should also be alert to the possibility of chronic subdural hematoma resulting from gait instability or falls, which are often associated with a history of alcoholism.

### 2.4.3. Frontotemporal dementia: Clinical signs mimic those of Alzheimer's disease, including functional urinary incontinence

Frontotemporal dementia (FTD), also called frontotemporal lobar degeneration, is a relatively uncommon cause of dementia, accounting for approximately 2.7% of all cases.<sup>37</sup> It encompasses several pathological subtypes, including TDP-43 proteinopathy, 3-repeat tauopathy (Pick's disease), and tau gene mutations. FTD is characterized by focal atrophy of the frontal and/or temporal lobes on MRI. In its early stages, patients may present with isolated behavioral disturbances, progressive non-fluent aphasia, or apraxia, corresponding to the location of lobar atrophy. FTD can also co-occur with motor neuron disease and may lead to OAB.

### 2.4.4. Progressive supranuclear palsy/corticobasal degeneration: Clinical signs mimic those of DLB, including OAB and fUI

Progressive supranuclear palsy (PSP; comprising 3.9% of Parkinsonism, with supranuclear eye-movement disorder sometimes occurring) and corticobasal degeneration (CBD; comprising 1.4% of Parkinsonism,<sup>38</sup> with alien hand, apraxia, and dystonic posture sometimes occurring) are relatively rare causes of gait and cognitive disorders. Pathologically, PSP and CBD share four-repeat tauopathy; therefore, their clinical features may occasionally overlap.

Autonomic dysfunction, including bladder dysfunction, has not traditionally been considered a feature in PSP/CBD. However, recent studies suggest that PSP/CBD can produce OAB, and to a much lesser extent, urinary retention.

### 2.4.5. Huntington's disease

Huntington's disease is a rare autosomal dominant disease due to the huntingtin (*HTT*) gene, in which the CAG repeat is markedly prolonged. Clinical features include chorea and dementia. Brain MRI shows atrophy of the caudate head and putamen.

Studies have reported that Huntington's disease patients exhibit OAB and detrusor overactivity, most probably due to brain striatal pathology.<sup>39,40</sup>

### 2.4.6. CADASIL/CARASIL: Clinical signs mimic those of WMD, including OAB and fUI

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL; *NOTCH3* gene mutation) and cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL; *HTRA1* gene mutation) are relatively recently recognized disease entities that simulate WMD, since the major pathology of these disorders is leukoencephalopathy. Clinical features of these disorders include OAB, gait difficulty, and dementia (executive dysfunction), presumably reflecting frontal pathology,<sup>41</sup> while the onset is considerably earlier than that of WMD.

### 2.4.7. Neuronal intranuclear inclusion disease: Clinical signs mimic those of AD, including fUI and OAB

Neuronal intranuclear inclusion disease is a rare cause of dementia due to the *NOTCH2NLC* gene, in which the CGG repeat is markedly prolonged. The subcortical lace sign is a characteristic MRI finding of this disorder. Neuronal intranuclear inclusion disease may also cause OAB.

### 2.4.8. Prion diseases: Clinical signs mimic those of Alzheimer's disease, including functional urinary incontinence

Prion disease, also called Creutzfeldt–Jakob disease, is a rare cause of dementia, but the disease progression is much faster than in AD or DLB. Creutzfeldt–Jakob disease may cause OAB, and rarely, urinary retention.

## 3. Overactive bladder and functional urinary incontinence: Brain mechanisms and management

Overactive bladder and fUI in older individuals are considered symptoms of the aging brain, often resulting from a combination of multiple pathologies, as described above. In general, management of OAB and fUI should be individualized, and the risk–benefit ratio of procedures, particularly invasive tests and treatments, must be carefully considered.

## 3.1. Overactive bladder: Reflecting frontal–basal ganglia pathology

### 3.1.1. Mechanism of overactive bladder

Overactive bladder, characterized by urinary urgency and frequency, is a major lower urinary tract symptom associated with brain diseases. Clinically, it is important to note that OAB can be the sole initial symptom of brain diseases such as WMD and NPH. Patients may visit urologists before the underlying etiology of OAB is confirmed. Therefore, collaboration between urologists and neurologists is recommended. Symptomatic patients commonly show detrusor overactivity (DO) on urodynamic testing. While OAB is considered a “primary” symptom of brain disease, the underlying mechanism by which brain disorders cause OAB or DO remains incompletely understood.

Experimentally, DO appearing after stroke in rats requires mRNA synthesis in the pontine micturition center of the brainstem. It is postulated that DO represents an exaggeration of the spino–bulbo–spinal (sacral cord–brainstem–sacral cord) micturition reflex that pathologically appears during bladder filling, whereas it normally functions during micturition. This exaggeration results from loss of central inhibition via the nucleus basalis of Meynert–prefrontal–cingulate–insular cortex cholinergic M1 and M3 pathways, and prefrontal–nigrostriatal dopaminergic D1 pathways.<sup>42</sup> Some patients with brain disease exhibit DO during bladder filling and detrusor underactivity (DU) during voiding on urodynamic testing (combined DO–DU). The origin of the DU component in these patients remains unclear. However, patients with brain diseases (such as WMD or DLB/PD) rarely show large post-void residuals.

### 3.1.2. Management of overactive bladder

Drugs used to treat dementia in AD can affect bladder function. Centrally acting acetylcholinesterase inhibitors, such as donepezil, are the primary treatment for managing cognitive symptoms. In some patients, donepezil can cause peripheral gastrointestinal side effects, including nausea and diarrhea, due to muscarinic M3 receptor stimulation. Its effects on bladder function, however, are more complex. Hashimoto *et al.*<sup>43</sup> reported that approximately 7% of patients taking 5 mg/day of donepezil experienced urinary incontinence. Sakakibara *et al.*<sup>44</sup> found that donepezil could augment DO, likely via peripheral muscarinic M3 stimulation, while also increasing bladder capacity, possibly through central mechanisms. In experimental animal studies, lesions in the nucleus basalis of Meynert—a central cholinergic nucleus projecting to the frontoparietal cortex—reduced bladder capacity. Central stimulation of muscarinic M1 and M3 receptors inhibited the micturition reflex, indicating that brain acetylcholine has a bladder-inhibitory role.<sup>45</sup> Furthermore,

improved cognition and alertness from donepezil may enhance a patient's initiative and ability to postpone urination.

Drugs used to treat gait disturbances in DLB/PD can also affect bladder function. A detailed clinical study showed that levodopa, which stimulates D1 and D2 receptors, exerts biphasic effects on OAB in PD patients—initially worsening symptoms over hours (“early” phase) and subsequently improving them over weeks (“late” phase).<sup>46</sup> In a rat model, a single dose of the dopamine receptor agonist apomorphine (stimulating both D1 and D2 receptors) similarly produced biphasic effects; high doses worsened bladder dysfunction, while low doses improved it. The mechanisms underlying these complex responses remain incompletely understood. Post-synaptic D1 (excitatory) and D2 (inhibitory) receptors have millimolar dopamine affinity, whereas dendritic D2 autoreceptors (inhibitory) have picomolar affinity. Thus, low-dose levodopa may initially stimulate dendritic D2 autoreceptors, suppressing dopaminergic neurons and facilitating the micturition reflex. In PD patients on long-term levodopa therapy, dopamine receptors may be downregulated, leading to potential hypersensitivity. Drugs targeting both D1 and D2 receptors are preferred over D2-selective agents, as the central D1 pathway is thought to inhibit the micturition reflex. The A11 dopaminergic cell group, located in the dorsal–posterior hypothalamus, is affected in MPTP-induced Parkinsonism in marmosets. This group provides the sole spinal dopamine input and may contribute to bladder overactivity. Peripheral D1 and D2 receptors are also present in the bladder, although their functional roles remain poorly studied. Clinically, the “late” ameliorative effect of levodopa on OAB can be expected in drug-naïve DLB/PD patients.

Behavioral therapy, such as bladder training, is suitable for older individuals with OAB or urge urinary incontinence who do not have severe dementia.<sup>47,48</sup>

Conversely, drugs used to treat OAB in AD can affect cognitive function. Anticholinergics are commonly used for neurogenic bladder dysfunction, especially in spinal cord injury, where they reduce high bladder pressure, vesicoureteral reflux, and urinary incontinence associated with combined DO–DU and detrusor–sphincter dyssynergia. In contrast, geriatric patients with AD and WMD typically exhibit OAB or DO alone, with minimal post-void residuals. Geriatric patients are particularly vulnerable to cognitive decline. If anticholinergics cross the blood–brain barrier (BBB), they can reach the brain and block central cholinergic receptors, especially M1 muscarinic receptors in the cerebral cortex and M4 receptors in the basal ganglia. Centrally acting anticholinergics, such as trihexyphenidyl (used for motor disorders in DLB/PD), worsen cognitive function in both experimental animals and humans. Similar effects have been reported with atropine (used during endoscopy or surgery) and

scopolamine (hyoscine, for colicky pain or motion sickness). Factors influencing these cognitive effects include: (i) high affinity for central muscarinic receptors, particularly M1; and (ii) ease of BBB penetration, determined by high lipid solubility ( $\text{LogP} > 3$ ), low hydrogen bonding ( $< 8$ ), low polarity (polar surface area  $< 90 \text{ \AA}^2$ ), neutral charge or low ionization, limited rotatable bonds ( $< 5$ ), and small molecular size ( $< 450 \text{ Da}$ ). Most bladder-targeted anticholinergics are non-selective muscarinic blockers, with darifenacin being an exception, as it is an M3-selective antagonist.

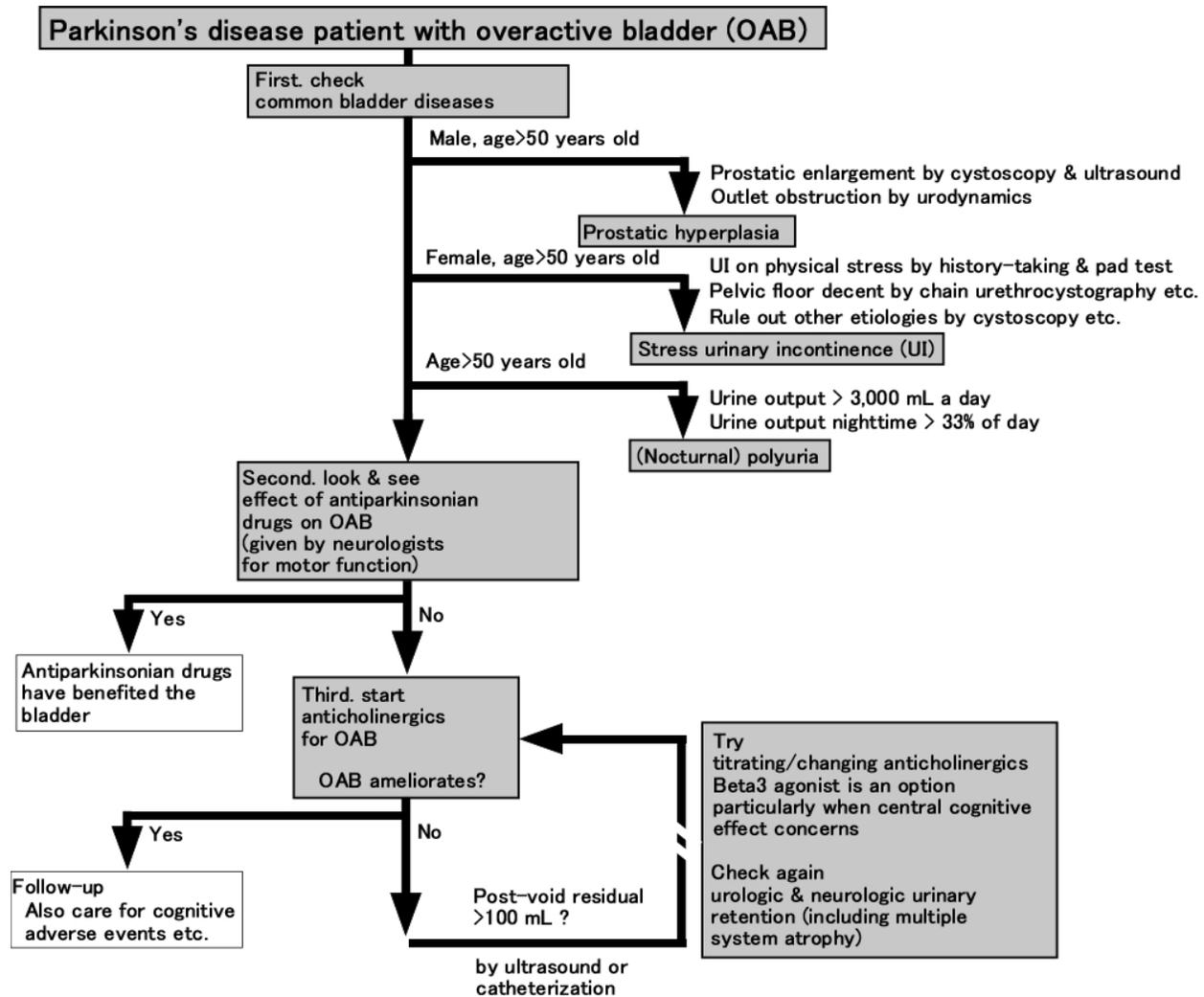
Regarding BBB penetration, most anticholinergics are 300–400 Da, but oxybutynin penetrates the BBB readily due to its high lipophilicity and neutrality, whereas trospium, a quaternary amine, has very high polarity and minimal central penetration. In summary, drugs that poorly penetrate the BBB are preferred for geriatric patients. When a central acetylcholinesterase inhibitor is already prescribed, a peripherally acting anticholinergic may be added cautiously. Sakakibara *et al.*<sup>44</sup> reported that in patients taking donepezil, careful addition of the peripherally acting anticholinergic imidafenacin did not worsen cognitive function as measured by the Alzheimer's Disease Assessment Scale–cognitive subscale.

More recently, selective  $\beta_3$  adrenergic receptor agonists (e.g., mirabegron, vibegron) are being recommended for the treatment of OAB in older individuals. This is because these drugs have not been associated with cognitive adverse effects, as assessed by the Montreal Cognitive Assessment.<sup>49</sup>

A summarized framework for initial OAB drug selection is shown in [Figure 3](#) and presented in [Table 1](#). In general, many geriatric patients may have a combination of AD (dementia) and WMD (OAB with gait difficulty); therefore, selective  $\beta_3$  adrenergic receptor agonists can be a good option because they do not interfere with cognition in AD. For pure AD patients with mild OAB, the same choice is recommended. For pure WMD patients, in addition to selective  $\beta_3$  adrenergic receptor agonists, anticholinergic drugs that do not easily penetrate the BBB can be started if OAB remains marked. For DLB (OAB with dementia and gait difficulty), the treatment follows that of AD. In addition, the above-mentioned dopaminergic drugs can be started for the management of gait difficulty.

If the above drugs do not work or are contraindicated, serotonergic drugs may be effective because the serotonergic pathway in the brain appears to be bladder-inhibitory.

Detrusor injection of botulinum toxin A is widely used to treat intractable OAB or urinary incontinence due to neurological diseases, including spinal cord injury.<sup>50</sup> However, in the geriatric population, this therapy should be applied with caution. Tibial nerve and other external stimulation therapies are performed; however, their effectiveness varies. Surgical



**Figure 3.** Flow chart for bladder management of neurologic patients (e.g., Parkinson's disease) with OAB. Geriatric patients commonly present with OAB. This flow chart applies to patients with established neurologic conditions. First, even though most bladder disorders in these patients may be caused by the brain disease itself, we check for common bladder diseases. Second, the effect of antiparkinsonian drugs on OAB (drugs given by neurologists for motor function) is assessed. If the OAB is ameliorated, then the antiparkinsonian drugs benefit bladder function. If not, the drug may not significantly change bladder conditions. Third, if necessary, the patient is started on anticholinergic drugs or selective  $\beta_3$  receptor agonists to reduce OAB. If OAB improves, we follow up on bladder function. We also monitor cognitive adverse events by asking caregivers and patients or performing a cognitive screening test, because anticholinergics may worsen cognitive function, particularly in elderly patients. Notably, post-void residuals (measured by ultrasound sonography or catheterization) may increase ( $>100$  mL) if OAB treatment does not produce significant effects. If post-void residuals increase, anticholinergic medication can be titrated or changed. In this case, we try to ascertain the urologic and neurologic causes of urinary retention, which can include multiple system atrophy. This image is original by the author. Abbreviations: OAB: Overactive bladder; UI: Urinary incontinence.

interventions, such as ileocystoplasty, bladder augmentation, and sacral neuromodulation, should be delayed in this population because of their invasiveness.

Finally, it is noteworthy that WMD in older individuals can present with OAB initially, and such patients may visit urologists first. For the correct diagnosis, to avoid unnecessary prostatic surgery, and to ensure appropriate patient care, collaboration between neurologists and urologists is important.

## 3.2. Functional urinary incontinence: Reflecting temporal-parietal or frontal-basal ganglia pathology

### 3.2.1. Mechanism of functional urinary incontinence

While OAB is a “primary,” often early brain symptom, fUI is considered a “secondary,” often late brain symptom. In general, fUI refers to incontinence without lower urinary tract pathology, arising secondary to immobility or falls (inability to reach the toilet; the major responsible sites are frontal-basal ganglia pathology), dementia (e.g., spatial disorientation; the

**Table 1. List of medications for overactive bladder treatment**

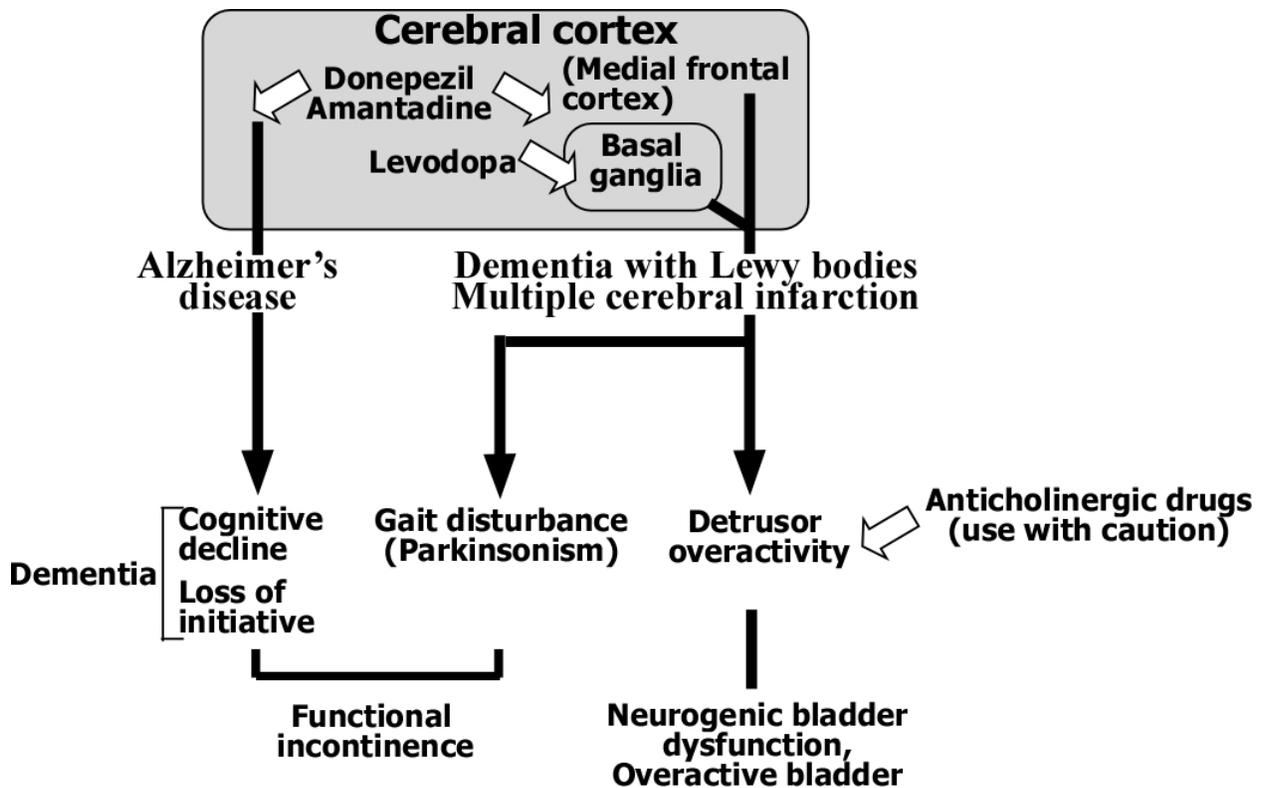
Medication	Main action	Potency	Adverse events: Systemic	Adverse events: Brain
Anticholinergics	Block peripheral ACh receptors to relax the bladder	Generally strong to moderate	Dry mouth, constipation (care should be taken for DLB with constipation)	Cognitive decline and delirium due to blocking brain ACh receptors
Oxybutynin	-	-	-	Can be present because it easily penetrates the blood–brain barrier
Tolterodine	-	-	-	Minimum
Fesoterodine	-	-	-	Minimum
Darifenacin	-	-	-	Minimum
Solifenacin	-	-	-	Minimum
Imidafenacin	-	-	-	Minimum
Trospium	-	-	-	Minimum
Selective beta-3 receptor agonists	Stimulate peripheral adrenergic $\beta$ 3 receptors to relax the bladder	Generally moderate	Few	No cognitive decline or delirium
Mirabegron	-	-	-	Absent
Vibegron	-	-	-	Absent

Abbreviations: ACh: Acetylcholine; DLB: dementia with Lewy bodies.

major responsible sites are temporal–parietal pathology), or loss of initiative (reduced motivation for toileting), often progressing to total dependence.<sup>51</sup>

As brain diseases advance, patients who cannot walk 3 meters without assistance or whose MMSE scores fall below 10/30 are likely to experience incontinence or require pads. Post-stroke patients may also develop incontinence due to disturbed consciousness or left hemispatial neglect, the latter

As brain diseases advance, patients who cannot walk 3



**Figure 4.** Relationship between functional urinary incontinence and overactive bladder. The image is original by the author.

reflecting lesions in the right hemisphere. The relationship between fUI and OAB is shown in [Figure 4](#).

In rehabilitation settings, urinary incontinence often correlates with larger brain lesions and predicts poorer outcomes on the Functional Independence Measure. These findings suggest that improving patients' initiative and mobility for toileting can reduce incontinence. Conversely, nocturia and OAB may lead to sleep deprivation, which negatively affects cognitive performance—such as cognitive slowing and memory impairment—and increases the risk of falls and fractures.

### 3.2.2. Management of functional urinary incontinence

Toileting and behavioral therapies are commonly used to manage fUI.<sup>52</sup> In prompted voiding, patients are asked at regular intervals whether they need assistance with toileting, and help is provided only if they request it. In scheduled toileting, patients are assisted on a fixed or individualized schedule, often every two hours. Prompted voiding is generally preferred, and selecting patients most likely to benefit from these regimens helps balance the cost of care with maintaining dryness.<sup>53</sup>

Enhanced visual cues—such as brightly colored toilet doors or large images of a person sitting on a toilet—can reduce incontinence in severely demented patients in psychogeriatric wards and nursing homes. Recommended environmental modifications include mobility aids (e.g., hallway handrails, canes, walkers, wheelchairs), easy access to and visibility of toilets, improved facilities (e.g., better lighting, grab bars, appropriate toilet seat height), automatic washing devices or lift-up commodes, and clothing designed for easier disrobing. Recent studies have shown that cholinergic medications can improve both cognitive function and OAB/fUI.<sup>54-56</sup>

## 3.3. Other causes and comorbidities

### 3.3.1. Transient causes of urinary incontinence

The first step in managing incontinence is to identify and address transient or acute causes. These causes can be remembered with the mnemonic “DIAPERS”: delirium, infection, atrophic vaginitis, pharmaceuticals, psychological factors, endocrine conditions, restricted mobility, and stool impaction.<sup>56</sup> Some of these factors arise from the underlying dementing illness, while others result from comorbid medical conditions, an inappropriate environment, or medications. Many of these factors are interrelated—for example, delirium in an older patient may be triggered by medication effects or an infection.

Medications can lead to various types of bladder dysfunction, with drug-induced urinary retention and overflow incontinence being particularly important. Causative drugs include antidepressants, serotonergic antipsychotics (used

for agitation, insomnia, or depression), anticholinergics (for colicky pain or OAB), and certain flu medications. Post-void residuals should be assessed using ultrasound or transurethral catheterization.

### 3.3.2. Stress urinary incontinence and outlet obstruction

Pelvic organ prolapse and stress urinary incontinence in women result from pelvic floor descent and weakness, most commonly affecting multiparous women over 50 years of age. Less invasive surgical repair is generally preferred for treating these conditions. Tricyclic antidepressants are frequently used to manage both OAB and stress urinary incontinence.  $\alpha$ -adrenergic agonists, such as midodrine hydrochloride, can strengthen the internal sphincter, while  $\beta$ 2-adrenergic agonists, such as clenbuterol, may enhance the external sphincter, providing benefit for some older women with stress incontinence. Pelvic floor exercises combined with biofeedback are also effective.

Benign prostatic hyperplasia (BPH) is common in older men. Outlet obstruction can cause not only poor urinary flow but also DO; DO associated with BPH is typically terminal, in contrast to the phasic DO seen in brain diseases. Surgery is considered for BPH when conservative measures or medications are insufficient or inappropriate. The proximal urethra contains a high density of  $\alpha$ 1A and  $\alpha$ 1D adrenergic receptors, whereas the vascular wall—especially in the elderly—predominantly expresses  $\alpha$ 1B receptors. Selective  $\alpha$ 1A/D-adrenergic blockers, such as tamsulosin and naftopidil, are preferred because they minimize side effects like postural hypotension.

### 3.3.3. Nocturnal polyuria

Diabetes is a common cause of polyuria. Nocturnal polyuria (nocturnal urine production >30–33% of total 24-hour urine output) mostly results from systemic atherosclerosis (e.g., ischemic cardiomyopathy, chronic heart failure, or chronic kidney disease) that leads to nocturnal rostral fluid shift. Another mechanism is a partial damage in the hypothalamic suprachiasmatic nucleus (circadian rhythm center) and/or the paraventricular nucleus (producing arginine vasopressin, an antidiuretic hormone that normally increases during nighttime). In DLB/PD, nocturnal polyuria is closely associated with detrusor overactivity, both of which are thought to be regulated by the dopaminergic pathways. Desmopressin is used to reverse nocturnal polyuria as a nasal spray or a tablet. Desmopressin also ameliorates orthostatic hypotension in the morning.

## 4. Conclusion

This review examines geriatrics and the bladder from a

neuropsychological perspective. Aging occurs predominantly in the brain. Among octogenarians (>80 years of age), WMD (80%), AD (33%), and DLB (8%) are common, and various combinations of these brain diseases may occur. WMD is thought to be the major pathological cause of early-onset OAB. In advanced AD or DLB cases, fUI may occur due to immobility or severe dementia. Appropriate management for both early OAB and advanced fUI is needed in the geriatric population, since these conditions are potentially treatable. This review highlights the unique relationship between aging brain pathology and urodynamic findings associated with fUI.

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The authors declare no conflicts of interest.

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