台灣良性攝護腺(前列腺)肥大症

臨床診療指引

Clinical Practice Guidelines for

Benign Prostatic Hyperplasia in Taiwan

目錄

指引

1. **指引的需要性**

近年來受惠於醫學之進步，人類壽命得以延長，但由於人口老化，而使得良性攝護腺肥大益受重視，更是健保給付前十大疾病之一。治療良性攝護腺肥大的相關文獻眾多，觀念更新的速度迅速，憑臨床醫師個人之力難以閱讀所有文獻。本指引所秉持的理念即是集合眾專家之力，分工合作，搜尋及閱讀所有相關文獻，將其做系統性的整理，從而歸納出實際可行的臨床建議及治療指引。

2. **指引發展歷程及版本增修狀態**

本指引爲國衛院第一版 “台灣良性攝護腺(前列腺)肥大症臨床診療指引”，其內容除了本土專家共識會議結論之外，並參酌美國泌尿科醫學學會和歐洲泌尿科醫學學會的良性攝護腺肥大臨床診療指引及台灣國家衛生研究院衛生政策研發中心實證臨床指引平台網站。提供以證據爲基礎的治療結果，讓醫師能提供病患做適當的治療。

3. **指引專業科別**

泌尿科、內科、家醫科

4. **指引範圍**

此指引適用於良性攝護腺肥大患者、第一線醫療從業人員以及提供各醫療院所之醫護人員診療參考。

5. **指引發展單位的聲明**

此指引的目的爲提供臨床醫師治療病患之參考，此指引並不提供任何形式之標準療法，亦不反對未被列入此指引的治療方式。依據此指引來治療病患並不能保證病患能得到良好的恢復。此指引的價值並不能取代臨床醫師的個人經驗，臨床醫師仍應依據個別病患的臨床狀況及臨床資料做出判斷，決定並採行對於個別病患最適合的治療方式。

6. **回顧與更新**

本指引預計於九十七年七月發表後定期進行回顧，並依據這段時間內新發表之文獻進行適度更新。未達回顧時間點，若有新文獻發表，且其證據強度可能足夠變更指引內容時，或學會已出版相關的更新指引時，則召開專家會議討論，取得是否更新及如何更新指引之共識。

7. 指引發展團隊

主持人 李良明 醫師　　共同主持人 陳光國 醫師

列席 台灣泌尿科醫學會 江漢聲 理事長、吳建志 秘書長

秘書 溫玉清 醫師

指引發展委員(依筆劃排列)

醫　院	科　別	姓　名
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成功大學附設醫院	泌尿部	唐一清 醫師
高雄榮民總醫院	泌尿外科	吳東霖 醫師
高雄醫學大學附設中和紀念醫院	泌尿科	黃書彬 醫師
高雄長庚醫院	泌尿科	徐淳建 醫師
國泰醫院	泌尿科	羅華安 醫師
馬偕醫院	泌尿科	許炯明 醫師

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新光醫院	泌尿科	黃一勝 醫師
臺北醫學大學市立萬芳醫院	泌尿科	李良明 醫師
臺北醫學大學附設醫院	泌尿科	葉劭德 醫師
慈濟花蓮醫院	泌尿科	郭漢崇 醫師
嘉義長庚醫院	泌尿科	陳志碩 醫師
彰化基督教醫院	泌尿外科	張進寶 醫師

8. 經費來源

國家衛生研究院衛生政策研究中心委託臺北醫學大學市立萬芳醫院統籌辦理

9. 參與指引發展之相關團體代表

台灣泌尿科醫學會、台灣尿失禁防治協會。

10. 財務與利益衝突聲明

與任何個人或團體無財務及利益之衝突。

方法學

1、文獻尋找

本指引之製定，於“台灣良性攝護腺(前列腺)肥大症臨床診療指引”共識小組會議中決定要討論之主題，其主題共分八項。包括了背景、危險因子、診斷與評估、治療建議、藥物治療、手術治療、另類治療、罕見或嚴重併發症的治療。撰稿人依主題搜尋 Medline 資料庫，自 1985 至 2007 之間的所有文獻，包括英文及中文文獻。納入條件爲臨床研究，排除條件包括了動物實驗、臨床技術敘述（technical note）、非英文及非中文文獻。搜尋文獻所使用之關鍵字及搜尋策略由個別撰稿人自行決定。

2、證據等級(Levels of Evidence)及建議強度(Grades of Recommendation)

參考中央健康保險局臨床診療指引發展手冊及國家衛生研究院實證臨床指引平台。

建議的形成是根據文獻的證據等級而來，建議強度與證據等級有關，但建議強度並非表示建議的重要性。證據等級分成八個等級，分類如下：

證據等級

等級	實證類別
1++	高品質之整合分析（Meta analysis），系統性文獻回顧（Systematic reviews）之隨機控制試驗（Randomized controlled trials, RCTs），或該隨機控制試驗之設計誤差（bias）極低。
1+	執行良好之整合分析，系統性文獻回顧之隨機對照試驗，或該隨機對照試驗之設計誤差極低。
1-	整合分析、系統性文獻回顧之隨機對照試驗，或該隨機對照試驗之設計誤差偏高。
2++	經過病例對照研究（case-control study）或世代研究（cohort study）之高品質系統性文獻回顧。 高品質的病例對照研究法及世代研究法可降低干擾、誤差及機率，並且具有高度的因果相關。
2+	經過病例對照研究或世代研究之設計良好的系統性文獻回顧。
2-	研究設計誤差較高之病例對照研究或世代研究。
3	非分析性之研究，例如：個案報告。
4	專家意見。

建議強度之評等

評等	內容
A	至少有一項隨機對照試驗之整合分析、系統性文獻回顧，或是隨機對照試驗之證據等級爲 1^{++}，且該研究可直接應用於目標群眾(target population)；或 由許多證據等級爲 1^{+}之統合分析，系統性文獻回顧(systematic reviews)或隨機對照試驗(RCTs)之研究構成的證據，可直接應用於目標群眾，且整體的證據有一致性的結果。
B	有許多證據主要由實證等級爲 2^{++}之研究構成，可直接應用於目標群眾，且整體而言證據有一致性的結果；或 從證據等級爲 1^{++}或 1^{+}研究證據所額外推論的證據。
C	證據主體由實證等級爲 2^{+}之研究構成，可直接應用於目標群眾，且整體而言證據有一致性的結果；或 從證據等級爲 2^{++}研究所額外推論的證據。
D	證據等級爲 3 或 4；或 從證據等級爲 2^{+}研究所額外推論的證據。
Ⅰ	相關證據不充足 (insufficient evidence)

背景

良性攝護腺肥大與老化有密切的關係〔1〕。雖然它本質並不是致命的疾病，但它的臨床症狀，如下泌尿道症狀(low urinary tract symptoms, LUTS)會影響病人的生活品質。由文獻報告，可知 65 歲以上的男性，有將近 30%的人被下泌尿道症狀所困擾。

1.盛行率

在過去 20 年，全世界各地都有許多良性攝護腺肥大的流行病學研究，但臨床上良性攝護腺肥大的盛行率仍舊無法確定。主要是良性攝護腺肥大在臨床上缺乏一個標準化的定義，因而難以進行適當的流行病學研究。在已發表的許多流行病學研究之中，有的是整個國家中的可能性樣本，有的是以年齡層來做隨機抽樣。有的是從一般(非專科)醫師、從醫院來的，有的是從篩檢問卷的回答者而來的。因爲使用的問卷不同以及研究的方式不一樣，因此良性攝護腺肥大缺乏一致性的盛行率。

Barry 等人根據年齡與攝護腺之組織學研究，提出了良性攝護腺肥大的組織學盛行率〔2〕。組織學上良性攝護腺肥大尚未發現在 30 歲之下的男性發生，但是機率隨著年齡上升而增加，在八十多歲到達顛峰(約 88%)〔2〕。大約有 20%的六十歲男性與 43%的八十歲男性可被檢查到肥大的攝護腺; 然而，肥大的攝護腺並非總是與臨床症狀有絕對的關係〔3〕。

臨床上，良性攝護腺肥大是一種高盛行率的疾病。在一項老化的縱向研究調查中發現，到了六十幾歲時，將近 60%之巴爾的摩(Baltimore)男性居民或多或少有某種程度的良性攝護腺肥大臨床症狀〔3〕。在美國 Olmsted 郡的 40-79 歲白種男性抽樣調查顯示，中度至重度的症狀可能發生在 13%的 40-49 歲男性以及在 28%的大於 70 歲男性〔1〕。在加拿大，針對 50 歲以上男性的一項電話調查，發現 23%的人有中度至重度的症狀。下泌尿道症狀的盛行率在歐洲與在美國大致是相似的，在蘇格蘭及荷蘭，症狀的盛行率從四十歲的 14%上升到六十歲的 43%〔4〕。由於樣本的選擇不同，中度至重度症狀的盛行率從法國的 14%到荷蘭的 30%都曾被報告過，而且發現中度至重度症狀的人口比例每多十歲就增加一倍。歐洲最近一項流行病學研究，以國際攝護腺症狀評分(international prostate symptom score, I-PSS)評估，大約 30%的 50-80 歲德國男性有中度至重度的症狀(即 I-PSS > 7)。在亞洲的一項多國多中心的研究顯示，中度至重度症狀的特定年齡(age-specific)百分比比在美國的高〔5〕，盛行率從四十多歲的 18%上升到七十多歲的 56%。有趣的是，對於兩個研究方法相似的美國人與日本人的報告，日本人的攝護腺平均重量似乎比美國人來得小〔6〕。總而言之，儘管方法學有所不同，以上所提到的研究可以有以下幾點結論：

(1) 輕微的下泌尿道症狀在 50 歲以上的男性是相當常見的。

(2) 輕微的症狀困擾較小，而中度和重度症狀則會造成較嚴重的生活困擾。

(3)相同程度的症狀可能有不同程度的困擾及生活品質的影響。
(4)臨床症狀、攝護腺大小及尿流速三者之間的關連性相對來講是低的。〔7〕
總之，良性攝護腺肥大仍然需要一個流行病學的定義，並確定它真正的發生率。

2. 良性攝護腺肥大是否爲進行中的疾病？

良性攝護腺肥大經常會合併下泌尿道症狀，若這些症狀不治療的話，可能會產生如急性尿滯留等的嚴重併發症。目前已有長期臨床研究證據顯示良性攝護腺肥大是一種進行中的疾病，某些病人進行的速度會比其他人快的多〔8〕。因此，良性攝護腺肥大引起的下泌尿道症狀的惡化可以紀錄爲下列生理指數改變而得知。

傳統上的生理指數有以下幾點：

・最大尿流速降低
・餘尿量增加
・攝護腺體積變大
・症狀分數惡化（增加）

此外，也可以使用一些指標，例如急性尿滯留或接受攝護腺手術的次數，尿路動力學指數的改變及疾病相關的生活品質惡化程度來評估其繼續進展程度。攝護腺特定抗原指數是目前的焦點，它似乎可以作爲以上各項指數惡化的良好預測因子。

3. 疾病進行的預測指標

支持疾病是進行中的最強證據來自 Olmsted 郡社群爲主的研究〔9〕及 Proscar longterm efficacy and safty study (PLESS) 安慰劑組的研究〔10〕。

作爲疾病進行預測指標的單一參數之證據強度總結於表一，並分類爲強，弱或無。文獻中決定之單一參數進行之確實速率顯示於表二。這些參數可能可以用於決定何種治療。可以針對有更明顯之疾病進行徵兆的病人給予預防性的措施。同樣的措施也可以應用於有高危險因子的病患。而疾病進行的危險因子包括有年齡，攝護腺特定抗原指數及攝護腺大小。還有其他幾種併發症例如腎功能受損及膀胱功能受損等，已被認爲和良性攝護腺肥大之進行有關。雖然這些相當重要，但卻很少出現，所以在社區爲主的臨床研究中較無法被精確的評估〔8〕。

表一：良性攝護腺肥大病情惡化指標之證據強度

	指標	社區爲主之研究	臨床試驗
下泌尿道症狀	IPSS	S	N/W
	BII	S	N/N
	生活品質	N	W/S
良性攝護腺體積變大	肛診	N	N
	經直腸超音波	S	S
	核磁共振	N	S/S
膀胱出口阻塞	最大尿流速	S	W/S
良性攝護腺肥大	病理組織	N/A	N/A
其他	急性尿滯留	S	S/S
	手術	S	W/S
	治療	S	N

S=強烈相關　W=弱相關　N=無證據顯示相關　N/A=無資料

IPSS=International prostate symptom score

BII=BPH Impact Index

表二：良性攝護腺肥大病情惡化個別指標

研究	進行速率						
	下泌尿道症狀	尿流速	攝護腺大小	急性尿滯留（每年一千人發生率）		手術（每年一千人發生率）	
				40-49 歲	大於 70 歲	40-49 歲	大於 70 歲
Olmsted	每年 0.18	每年-2%	每年 1.9%	3.0	34.7	0.3	10.9
Health Profressional	NR	NR	NR	3.3	11.3	NR	NR
PLESS	四年 -1.3	四年 +0.2mL/s	四年 +14%	四年 7%		四年 10%	
兩年研究	NR	NR	NR	1.6-4.2% 0.5-3.9%		NR	
北美	NR	NR	NR	NR		10-39%	

NR: not reported, PLESS: proscar longterm efficacy and safty study

危險因子

研究並了解良性攝護腺肥大的危險因子，將有助於預防良性攝護腺肥大的發生及減緩其疾病的進展。

1. 種族

種族本身似乎並不是導致良性攝護腺肥大的危險因子，因爲從各國男性死後解剖的研究發現：良性攝護腺肥大好發於四十歲以後的男性，不同國家、不同種族的男性在一定年紀時，其盛行率大致相似，而八十歲以上的男性則約 90%以上會有良性攝護腺肥大〔11,12〕。相對地，也有研究指出雖然不同種族的男性在組織學上良性攝護腺肥大的發生率大致相似，但症狀較嚴重良性攝護腺肥大的發生機率，美國黑人高於白人，而亞裔男性則是最低〔11 ~ 13〕。

2. 社會經濟地位

有研究報告社會經濟地位較高的男性較易發生良性攝護腺肥大〔14〕；另一方面卻也有研究指出社會經濟地位較低的男性接受攝護腺切除手術的比例較高〔15〕。這兩種截然不同的發現，或許只說明了一種可能，即社會經濟地位本身可能不是影響良性攝護腺肥大發生的危險因子，但它也許會影響到患者的就醫習慣及對治療療效的期待〔16 ~18〕。

3. 性生活

雖然早期有研究報告指出性生活頻率的多寡會影響良性攝護腺肥大的發生〔19〕，但後來許多類似的研究卻無法証實原先的發現。相對地，有越來越多的証據顯示良性攝護腺肥大所引起的排尿症狀可能影響到男性的性功能〔20, 21〕。

4. 輸精管結紮

輸精管結紮與否並不會影響到良性攝護腺肥大的發生及攝護腺體積的大小〔22〕。

5. 飲酒

過度飲酒的男性理論上會因體內血清中雌性激素濃度的增加、睪固酮濃度的降低和睪固酮代謝的增快〔23〕而較不會發生良性攝護腺肥大。但一些相關研究的結果未能完全証實此一理論〔24 ~27〕。大致上飲酒與良性攝護腺肥大的發生之間並無直接相關。

6. 肝硬化

一些研究發現肝硬化的男性會因體內荷爾蒙環境的改變而降低良性攝護腺肥大的發生可能〔18,19〕，即肝硬化男性的攝護腺肥大體積通常小於同年齡的一般男性〔28〕。

7. 高血壓

由於攝護腺和血管內同樣具有很多甲型腎上腺素的受體，因此理論上，血壓較高的男性，由於其體內受到較多腎上腺素的刺激，此時如果同時患有良性攝護腺肥大，則其排尿症狀應該也會較爲嚴重才對。雖有一些研究發現高血壓同時有良性攝護腺肥大，會發

生較明顯排尿症狀的比例確實較高〔27〕，然而其他的臨床研究結果並未能得到同樣的結論〔15, 30〕。又除了高血壓之外，心臟病患者似乎也比沒有心臟病的男性更易出現排尿症狀〔27〕。

8. 抽煙

由於抽煙者所吸入的尼古丁會影響男性血清中睪固酮和雌性激素的濃度，所以理論上抽煙應該會與良性攝護腺肥大的發生和發展有關。一些臨床研究發現抽煙者的攝護腺體積相對地較小，也較不易因良性攝護腺肥大而接受手術〔31, 32〕。相對地，也有研究發現目前或過去抽煙者較易發生程度較嚴重的排尿症狀〔27〕。而其他的一些臨床研究則發現抽煙與良性攝護腺肥大的發生或因良性攝護腺肥大而接受手術之間，並無一定相關〔15, 33〕。總體來說，如果抽煙真的與良性攝護腺肥大有關的話，其相關性也是相當微弱，而不具明顯的臨床意義。

9. 飲食

有研究發現食用較多的牛肉者的接受攝護腺切除率較高〔34〕。也有研究發現食用較多牛奶、奶油、人造奶油及較少食用黃、綠色蔬菜者，較易發生良性攝護腺肥大〔14, 35〕。但總括來說，目前尚無足夠的証據可以証明飲食與良性攝護腺肥大之間的相關性。

10.肥胖與身體質量指數

雖然原因仍不明，但已有許多証據顯示肥胖及身體質量指數過高的男性較可能發生良性攝護腺肥大，其排尿症狀較嚴重且接受攝護腺切除手術的機會較高〔32, 36, 37〕。除了肥胖之外，血糖過高及高密度脂蛋白膽固醇過低的男性也較容易發生良性攝護腺肥大〔27, 38〕。

直至目前爲止，良性攝護腺肥大危險因子的研究方法並沒有一定的準則可以遵循，不同研究的對象也常不同，因此其研究結果很難可以加以比較；同一目的的不同研究也可能令人不解地得到不同結果。大體而言，目前可以確定的是：年齡的增長和男性荷爾蒙的刺激是導致良性攝護腺肥大發生和進展的兩大因子。其他的危險因子如種族、生活飲食習慣等雖曾被廣泛研究，然其與良性攝護腺肥大之間的關係，現今仍有許多未明之處。

診斷與評估

1. 病史詢問（Clinical history）**（建議等級：A）**

對於具有下泌尿道症狀的男性患者而言，判斷其症狀是否由良性攝護腺肥大所造成的是相當重要的；因此，對於此類病患在開始進行評估時，必須進行詳細的病史詢問，這些病史必須特別著重於與泌尿系統可能有關的疾病上，尤其是一些會造成膀胱功能變化或是尿量增加的疾病。

2. 症狀評估（Symptom assessment）**（建議等級：A）**

對於良性攝護腺肥大症狀嚴重度的評估最好的方法是利用症狀評量表，目前最被廣爲使用的評量表是國際攝護腺症狀評分或美國泌尿科醫學會症狀指標(American Urological Association Symptom Index, AUA Symptom Index)，這兩個量表是相同的，有 7 個與攝護腺肥大症狀有關的問題，分數分別由 0 分到 5 分，將每一問題的分數加總，若總分數在 0-7 分，則爲輕度症狀，8-19 分爲中度症狀，20-35 分爲重度症狀；國際攝護腺症狀評分除了 7 個問題之外，另一個問題是關於患者對於良性攝護腺肥大影響生活品質的情形，評分範圍由 0 分（代表快樂的，delighted）到 6 分（代表糟透了，terrible）。利用這個量表，我們可以了解病人良性攝護腺肥大症狀的嚴重度，藉以決定治療的方向，並評估治療的效果。

3. 理學檢查（Physical examination）**（建議等級：A）**

理學檢查主要是重點式的神經學檢查及肛門指診（digital rectal examination, DRE），神經學檢查的重點在於評估整體精神狀態（mental status）、活動狀態（ambulatory status）、下肢肌肉神經功能及肛門括約肌張力。肛門指診主要目的有二：（1）檢查是否有攝護腺癌：局部擴散性的攝護腺癌也會出現下泌尿道症狀，肛門指診可以排除此種狀況。（2）評估攝護腺大小：攝護腺大小對於治療方式的選擇是很重要的因素，但是用肛門指診評估攝護腺大小常有低估的情形〔39, 40〕。

4. 檢驗室檢查（Laboratory examinations）

A. 尿液分析（Urinalysis）**（建議等級：A）**

膀胱結石、膀胱癌、尿路感染、尿道狹窄等狀況也都會出現下泌尿道症狀，雖然這些狀況不一定會出現異常的尿液分析結果，但是一個正常的尿液分析結果會使上述情形的可能性降低。

B. 血清肌酸酐（Serum creatinine）**（建議等級：B）**

攝護腺肥大患者中腎功能不良的比率並不會比同年齡的一般男性高，綜合數個大規模的攝護腺肥大臨床研究結果顯示，良性攝護腺肥大患者中具有腎功能不良的比率低於 1%，而且其中大部份不是良性攝護腺肥大所引起的腎功能異常。

C. 血清攝護腺特定抗原**（建議等級：A）**

攝護腺特定抗原的測量對於良性攝護腺肥大患者的評估具有幾個意義：（1）攝護腺特定抗原與年齡及攝護腺大小均有相關〔41~43〕，因此攝護腺特定抗原高代表攝護腺可能比較大。（2）攝護腺特定抗原高表示攝護腺癌的機會也比較大。（3）攝護腺特定抗原可做爲良性攝護腺肥大病程進展的指標，具有較高攝護腺特定抗原的良性攝護腺肥大患者表示其攝護腺成長、症狀及尿流速惡化、急性尿滯留及未來接受良性攝護腺肥大手術的機會均較高〔44~46〕。因此，測量攝護腺特定抗原對良性攝護腺肥大患者是極爲重要的。一般建議男性五十歲以上，有攝護腺癌家族史者四十五歲以上應接受攝護腺特定抗原檢查。

D. 尿液細胞學（Urine cytology）（**建議等級：B**）

對於以刺激性症狀爲主的患者，尤其是有抽煙史或是具有其他膀胱癌危險因子者，尿液細胞學檢查應加以考慮。

5. 尿流速檢查（Urinary flow rate, Uroflowmetry）（**建議等級：A**）

尿流速檢查一般建議至少進行兩次，且尿量至少要 150 毫升以上才具有參考價值。尿流速中的最大尿流速能據以預測手術的效果，若最大尿流速低於 10 毫升/秒，則比較可能有膀胱出口阻塞，手術的效果也會比較好；但尿流速並無法預測其他治療方式的效果。此外，一個具有明顯下泌尿道症狀但尿流速正常的患者，其症狀可能與良性攝護腺肥大沒有相關。

6. 排尿後餘尿（Post-void residual urine, PVR urine）（**建議等級：B**）

排尿後餘尿量測量可用單次導尿或腹部超音波測量；排尿後餘尿量太大（大於 200-300 毫升）代表患者可能有膀胱功能障礙，對於手術治療的效果可能較差。

7. 尿路動力學檢查（Urodynamic study）（**建議等級：B**）

尿流速慢只能表示患者可能有膀胱出口阻塞，要辨別尿流速慢是導因於膀胱出口阻塞或是膀胱收縮力不佳，必須仰賴更進一步的尿路動力學檢查；目前公認較佳的是膀胱壓-尿流速檢查 〔47~49〕，這是一個侵入性的檢查，因此必須視個案所須才安排。

儘管膀胱壓-尿流速檢查可以準確的診斷膀胱出口阻塞，對於高逼尿肌壓力且低尿流速的患者，可以預期手術會有良好的效果，但是它並無法預測藥物治療的效果。

8. 泌尿道影像學檢查（**建議等級：B**）

包含靜脈注射尿路攝影（intravenous urography, IVU）及超音波，可藉以檢查腎臟水腫、腎臟腫瘤、輸尿管腫瘤、膀胱腫瘤等，目前超音波被普遍認爲是較恰當的檢查工具，因其可以較清楚的檢查腎臟實質、攝護腺及膀胱等器官，對攝護腺大小、形狀的評估可利用腹部超音波或經直腸超音波。超音波檢查不具放射性、微侵入性、成本低，故被廣爲使用。

9. 尿道膀胱鏡檢查（urethrocystoscopy）（**建議等級：B**）

尿道膀胱鏡檢查可以檢查尿道、評估攝護腺大小、尿道阻塞程度及膀胱頸通暢程度等；

但因其侵入性較高，故必須選擇性的使用。

10. 排尿日誌紀錄（Voiding diary）**（建議等級：B）**

排尿日誌的記錄對於了解病患解尿型態具有很大的幫助，通常記錄 24 小時即已足夠〔50〕。排尿日誌對於頻尿與夜尿的評估尤其有幫助，例如夜尿患者中，夜間多尿症（nocturnal polyuria）的病人即可藉此診斷〔51~53〕。

治療建議

在 20 世紀的前半期，二種方法可應用於治療良性攝護腺肥大，一種爲敞開式攝護腺切除手術(Open prostatectomy)，另一種爲經尿道攝護腺切除術（Transurethral resection of the prostate, TURP），兩者皆爲外科手術方式。現今有許多不同的治療方法可供選擇，包括有藥物治療、微侵犯性手術治療等等，可根據不同病人，不同嚴重程度的症狀作選擇，以下將對各種不同的治療方法做一簡單說明。

1. 追蹤觀察(Watchful waiting)

追蹤觀察適用於輕度症狀的病人。對於中等或重度，但沒有因良性攝護腺肥大所引起的併發症時（例如：腎功能異常、尿滯留或反覆尿道感染）也可以。但無法預估何時會發生併發症？所謂追蹤觀察是病人經由醫師定期評估其症狀的嚴重程度，但不採取任何積極的治療。通常追蹤觀察是每年對病人作一次例行性的攝護腺檢查。之後根據檢查的結果來決定繼續追蹤觀察，或採取更積極的治療。

2. 藥物治療

包括有單一療法：甲型腎上腺素阻斷劑 (alpha-adrenergic blockers)，5-α 還原酶抑制劑 (5 alpha-reductase inhibitors)。合併療法：甲型腎上腺素阻斷劑合併 5-α 還原酶抑制劑。雖然良性攝護腺肥大症之藥物療效不如手術，但可以在較少及較輕微的副作用下，提供適當的症狀緩解〔54, 55〕。

A、甲型腎上腺素阻斷劑

選擇性的甲型-1 腎上腺素阻斷劑 如 alfuzosin, doxazosin, tamsulosin 與 terazosin 都是治療良性攝護腺肥大所導致的下泌尿道症狀的適當藥物。雖然這些藥物在副作用有些許不同，然而臨床療效近似。 [Agency for Health Care Policy and Research of the United States Department of Health and Human Services (AHCPR)專家意見]

(1)作用機轉：一般認爲甲型腎上腺素阻斷劑的作用機轉是經由抑制攝護腺和膀胱頸平滑肌收縮，而達到減低膀胱出口動態性阻塞的結果〔56, 57〕。然而各亞屬甲型腎上腺素受體的實際貢獻，以及這些藥物在生物體中對中樞神經的作用至今仍未明。尿路動力學檢查結果顯示，雖然和安慰劑相比，甲型腎上腺素阻斷劑確實可以改善尿流速度，然膀胱內壓力測定結果顯示出口阻塞的程度並未獲得改善。

(2)臨床效益：Djavan 和 Marberger 所做的 meta-analysis 顯示，和安慰劑相比，甲型腎上腺素阻斷劑可以改善整體症狀約 30-40%，尿流速度進步約 16-25%〔58〕。服用藥物後 48 小時之內患者就可以感受到症狀改善，如果超過一個月，症狀仍未獲

得改善就沒有理由繼續使用下去。文獻回顧分析顯示 alfuzosin，doxazosin, tamsulosin 及 terazosin 在症狀緩解的效果是相似的〔59〕。療效不明顯的患者可以考慮逐步調高劑量，但需注意可能之副作用。

(3)耐久性（durability）：有關長期使用甲型腎上腺素阻斷劑對良性攝護腺肥大症自然病史影響的大型研究目前仍然闕如。

(4)副作用：甲型腎上腺素阻斷劑主要被報告的副作用爲姿勢性低血壓，頭暈，疲倦，射精障礙及鼻塞〔58〕。藥物副作用在不同亞屬之甲型腎上腺素阻斷劑上有些許的不同，例如: tamsulosin 有較少機率的姿勢性低血壓, 但有較高的射精障礙機率。更大型，設計良好，直接比較的研究是需要的，才能斷言何者有較高的安全性。另有些研究指出，對於高血壓患者同時存有心臟危險因子時，單獨使用 doxazosin 比使用其他降血壓藥物有較大可能會引發鬱血性心衰竭〔60〕。因此當高血壓患者因下泌尿道症狀而接受 doxazosin 治療時，需提醒患者仍須尋求心臟科醫師建議控制其血壓。

B、5α 還原酶抑制劑

5α 還原酶抑制劑，finasteride 與 dutasteride 也可用於治療對良性攝護腺肥大症所導致的下泌尿道症狀，特別是攝護腺體積較大的患者。然而醫師在做出此建議前必須就個別病患情形予以評估其必要性和合理性。

(1) 臨床效益：5α 還原酶抑制劑對良性攝護腺肥大症是有其療效。它可能使攝護腺體積縮小 20-30%，症狀改善約 15%，尿流速度進步約 1.3-1.6 毫升/秒〔61 ~ 64〕。綜合六個隨機臨床試驗的統計分析顯示，當攝護腺體積超過 40 毫升時 finasteride 有較好的治療效果〔65〕。Finasteride 可以顯著減少良性攝護腺肥大症患者發生急性尿滯留或需要接受攝護腺手術的可能性〔66~ 68〕。治療前血清攝護腺特定抗原值大於 1.4 ng/ml 的患者會有較佳的長期療效〔69〕。多項研究發現，finasteride 在開始用藥六個月後可達最大功效，只要持續使用，它對於縮小攝護腺體積、改善主觀症狀和尿流速度的功效可以持續 5-10 年〔70 ~ 72〕。相較於甲型腎上腺素阻斷劑，5-α 還原酶抑制劑在改善下泌尿道症狀的效果較緩慢且較不顯著。（AHCPR 專家意見）。

Dutasteride 是一種新型的 5-α 還原酶抑制劑，可以同時抑制第一型和第二型 5-α 還原酶。Finasteride 可使血中雙水睪固酮 dihydrotestosterone (DHT)濃度減少 70%，dutasteride 則可減少達 90%〔73, 74〕。有 4 個大型的隨機雙盲臨床試驗發現 dutasteride 可以使攝護腺體積縮小 26%，改善症狀和尿流速度，同時減少攝護腺肥大症患者發生急性尿滯留或需要接受攝護腺手術的機率〔75, 76〕。

(2) 血尿：finasteride 可以被用來治療良性攝護腺肥大症所伴隨的血尿〔77, 78〕。

(3) 副作用：根據 PLESS 研究，常見的副作用有性慾減低（6.4%），勃起功能障礙（8.1%），射精量減少（3.7%），不到 1%的患者有皮膚疹、乳房腫脹、疼痛；但是

這些都是可逆的，且在第一年後就較少發生〔70, 79〕。另一項研究顯示 finasteride 連續使用 4 年不會影響骨密度〔80〕。

(4) 對攝護腺特定抗原的影響：每天服用 finasteride 5 mg 達 12 個月將使血清攝護腺特定抗原濃度減低一半，對於使用 finasteride 的患者只要將測得的攝護腺特定抗原濃度乘以 2 即可正確判讀，不致影響攝護腺癌偵測〔81 ~ 83〕。有關 finasteride 對游離攝護腺特定抗原的影響研究結果莫衷一是，但多數學者認爲游離的攝護腺特定抗原的比例受影響不大〔84, 85〕。

C、合併治療 (Combination therapy)

對明顯的良性攝護腺肥大症所造成的下泌尿道症狀，合併使用甲型腎上腺素阻斷劑與 5α 還原酶抑制劑可能是可接受的治療 (AHCPR 共識)。

一項爲期 5 年的研究報告(Medical Therapy of Prostatic Symptoms, MTOPS)顯示, 合併使用甲型腎上腺素阻斷劑與 5-α 還原酶抑制劑療法比單獨使用其中任一種藥物，在長期追蹤觀察中能更有效的減少發生急性尿滯留及需要接受良性攝護腺肥大相關手術的可能性〔86〕。接受 doxazosin 的患者和安慰劑組相比，發生症狀惡化的危險性降低了 39%, 在 finasteride 組是 34%, 而在接受合併治療的病患則足足降低了 67%。發生尿滯留的危險性，在 doxazosin 組減少 31%，finasteride 組減少 67%，合併治療組則減少 79%。需要接受攝護腺手術的可能性在 finasteride 組降低了 64%，合併治療組降低 67%，但單獨接受 doxazosin 治療的患者和安慰劑組相比並沒有改變手術危險性〔86〕。 然而合併治療法大幅增加了病人與醫療照護體系的負擔，所以是否值得這樣做，醫師需針對每個患者個別情況加以評估。一般而言，攝護腺越大、攝護腺特定抗原較高的病患，發生病情及症狀惡化的危險性越高；這些患者是最可能因合併治療而獲益的族群。

另一項研究則發現，部分良性攝護腺肥大的患者，在使用合併治療法 9-12 個月後，停止給予甲型腎上腺素阻斷劑並不會使原先已改善的下泌尿道症狀再度惡化〔87〕。SMART [Symptom Management After Reducing Therapy] 研究也發現合併使用 dutasteride 和 tamsulosin 的患者在停用 tamsulosin 後也不會有症狀惡化的情形〔88〕。

D、抗膽鹼劑(anticholinergics)：

接近五成的攝護腺肥大症患者同時有膀胱儲尿功能減退的困擾〔89〕，這些刺激性症狀對於病患造成的困擾事實上不亞於阻塞性症狀。理論上抗膽鹼劑藥物會使良性攝護腺肥大症患者餘尿量增加，甚至造成急性尿滯留〔90〕，因此美國及歐洲泌尿科醫學會都不建議良性攝護腺肥大症患者使用抗膽鹼劑藥物，不過臨床上有不少醫師在使用。有關這方面的研究並不多，Blake-James 等人〔91〕分析了 5 個隨機、對照臨床試驗和 15 個觀察性的試驗發現：抗膽鹼劑藥物可以增加膀胱容量，增加膀胱首度收縮

感時的尿量，進而減少患者頻尿次數，改善生活品質。病患可能自覺排尿稍微困難，但最大尿流速度變化不大，餘尿量有顯著增加但不具臨床意義，發生急性尿滯留的比率也不比對照組高〔92~94〕。不過這些研究因所收納的試驗對象人數仍不夠多，追蹤期間不夠長，無法知道長期使用對於病程發展是否會有影響。目前初步的結論是抗膽鹼劑藥物可以改善某些攝護腺肥大症患者的刺激性症狀，但最好與甲型腎上腺素阻斷劑併用，也不宜用在高餘尿量（定義爲 50 或 150cc）的患者〔91, 95〕。

D、結論（**建議等級：A**）

(1). 甲型腎上腺素阻斷劑可以迅速、有效改善良性攝護腺肥大所導致的下泌尿道症狀，和尿流速度。

(2). 一般而言 Alfuzosin、 doxazosin、 tamsulosin 和 terazosin 在療效和安全性方面並無明顯差異。

(3). 5-α 還原酶抑制劑可以縮小攝護腺體積，進而改善下泌尿道症狀，和尿流速度；其最大療效大約需 6 個月才能顯見。

(4). 5-α 還原酶抑制劑對於攝護腺體積較小的患者幫助不大。

(5). 5-α 還原酶抑制劑可以改變良性攝護腺肥大症病程進展，減低發生尿滯留或需行攝護腺肥大相關手術的可能性，然其成本效益有待評估。

(6). 只要適當判讀血清攝護腺特定抗原值（乘以 2），5-α 還原酶抑制劑不至於影響攝護腺癌偵測。

(7). 一般而言，攝護腺越大、攝護腺特定抗原較高的病患，發生病情及症狀惡化的危險性越高；這些患者最可能因合併療法而獲益。然目前仍無可靠指標可用以選擇適當患者。

3. 微侵入性手術

良性攝護腺肥大之熱療法(thermotherapy)是以高溫造成攝護腺組織的壞死(necrosis), 而達到和經尿道攝護腺切除手術類似的治療效果。微波(microwave)、高週波射頻(radiofrequency)及高能聚焦式超音波(high-intensity ultrasound)均可達到相同目的〔96~ 98〕。臨床試驗顯示溫度必需超過 45℃~50℃，才可以造成組織凝固，對良性攝護腺肥大的症狀才可能有改善〔98~ 100〕。

一、經尿道微波熱療法(Transurethal microwave thermotherapy, TUMT)

最適合接受經尿道微波熱療法治療的病人是有著中度到重度的膀胱出口阻塞, 並且合併有明顯攝護腺肥大〔101〕。術後導尿管放置的時間約一至二星期，會陰部位疼痛與急尿的症狀可能持續幾個星期〔102, 103〕。高能量經尿道微波熱療法與經尿道攝護腺切除手術相比,在一年的追蹤期後, 兩者對主觀症狀和尿流速改善的程度是相似的〔104〕。術後平均追蹤 2.5 年經尿道微波熱療法，其滿意度分別爲 82%，再手術率分

別爲 7.3%，國際攝護腺症狀評分改善 13 分，而最大尿流速從 10.5 至 15.5 毫升/秒。逆行性射精的機率約 44 %〔102~104〕。

美國食品與藥物管理局對於經尿道攝護腺熱療法有所顧慮，其建議：

1. 需確認病患符合熱治療的適應症，包括合適的攝護腺體積。需確定病患先前沒有接受過攝護腺或骨盆腔的放射治療，以避免直腸廔管形成的危險性。另外熱治療裝置對攝護腺癌患者的安全性與效用是未知的。
2. 熱療法需要醫師全程的監控，醫師需要: (1) 確認導尿管固定氣球(retension balloon)與直腸溫度感應氣球沒有滲漏, (2) 確認導尿管與肛門溫度感應裝置在治療前是裝置妥當的。
3. 治療時不建議使用全身或脊髓麻醉。如果病人感到過度的疼痛或異常，治療需暫時停止。

二、經尿道攝護腺針刺去除術（Transurethral needle ablation, TUNA）

經尿道攝護腺針刺去除術是另一種熱治療治療方式，可能以選擇在局部麻醉操作。在膀胱鏡目視下將細針穿過尿道扎到攝護腺組織，導入高週波，將攝護腺組織加熱到 100℃而產生組織壞死〔105〕。最理想的病人爲有阻塞性良性攝護腺肥大，攝護腺重量在 60 克以下且主要是雙側側葉肥大的病人〔106, 107〕。手術後尿滯留發生率約 13.3~41.6%，一星期內 90-95% 病人可移除導尿管〔105〕。非隨機性的臨床試驗都顯示經尿道攝護腺針刺去除術對症狀的改善率約 40-70%〔106 ~ 109〕。持續 4-6 週的刺激性排尿症狀是常發生的〔110〕。沒有令人信服的證據顯示攝護腺體積在接受經尿道攝護腺針刺燒灼術後有顯著減小〔107 ~ 113〕。有關長期療效的證據仍然有限〔114, 115〕。經尿道攝護腺針刺去除術對攝護腺體積超過 75 毫升的病人是不適合的。

三、經直腸高能聚焦式超音波 (Transrectal high-intensity focused ultrasound, HIFU):

利用高能聚焦的超音波將特定範圍內的攝護腺組織破壞。需要在全身麻醉或較深的靜脈鎮靜下施行，可改善排尿症狀 50-60%, 最大尿流速可改善 40~50%。長期的效果不明。目前尚未有隨機, 控制組的臨床試驗証實其療效。最顯著的副作用是較長時間的尿滯留, 約 3-6 天。在性生活活躍的男性，精血症發生率可高達 80%。逆行性射精與勃起功能異常是可安全地避免的〔116, 117〕。但確實療效目前仍在評估中。

建議 **(建議等級: B)**

經尿道微波熱療法、經尿道攝護腺針刺燒灼術及經直腸高能聚焦式超音波不建議當作第一線治療方式，但可作爲不適於手術(高危險性或不希望長期藥物治療)患者的另一種選擇。

4. 手術治療

當良性攝護腺肥大引起以下嚴重併發症時，一般而言會建議手術治療〔47〕：

1. 頑固性尿滯留(refractory retention)
2. 良性攝護腺肥大合併膀胱結石
3. 反覆性泌尿道感染
4. 反覆性明顯血尿
5. 良性攝護腺肥大引起阻塞性腎病變
6. 病人拒絕服藥
7. 藥物治療無效

手術治療方式如下：

A. 經尿道攝護腺切除術

儘管良性攝護腺肥大的手術治療方式層出不窮，目前經尿道攝護腺切除術仍被視爲攝護腺肥大的指標性手術。到目前爲止其他新的手術與經尿道攝護腺切除術的評估比較，其結果均無法優於經尿道攝護腺切除術；而且經尿道攝護腺切除術於長期追蹤之隨機臨床試驗中也已被證實其療效〔118〕。

經尿道攝護腺切除術乃利用經過尿道之內視鏡，藉助導電之鐵環（electrified loop）切除增生之攝護腺組織並同時以電燒止血。The Veterans Affairs Cooperative Study 證實此手術 1.4% 的尿失禁風險(發生率與觀察組相似)；其對性功能之影響也與觀察組一樣，分別約有 20% 的病患認爲其性功能變差；追蹤三年手術失敗率小於 10%〔119〕。經尿道攝護腺切除術之治療一般需用全身或半身麻醉，也需要住院。此項手術有一獨特的手術併發症，即所謂的經尿道攝護腺切除症候群(TURP Syndrome)，發生率約爲 2%〔120, 121〕。至於其他併發症包括：性功能障礙(3.4–32%)、逆行性射精(53 -75%)、膀胱頸攣縮(0.3-9.2%)、尿道狹窄(2.2-9.8%)、術中或術後需要輸血(0.4-7.1%)、尿路感染(1.7-14.0%)和術後出血形成血塊引起尿滯留(2-5%)等〔122〕。

建議(建議等級: A)

經尿道攝護腺切除術目前仍爲治療良性攝護腺肥大最有效之方法，但有較高比例會導致逆行性射精或無法射精，所以不建議施用於年輕病患或仍想保有授孕能力之病患。

B. 經尿道攝護腺切開術(Transurethral incision of the prostate, TUIP)

經尿道攝護腺切開術是一項以全身麻醉或神經阻斷之局部麻醉於門診即可施行的內視鏡手術，但建議使用於較小之攝護腺(一般若可切除之攝護腺重量小於或等於三十公克可建議施行)。如果適當的選擇病人，此項手術對於病人症狀之改善是與接受經尿道攝護腺切除術者相當的〔123 ~ 126〕。與經尿道攝護腺切除術的隨機控制試驗比較，此

項手術可降低發生逆行性射精的風險(18.2% vs. 65.4%)，也會減少輸血的機會(0.4% vs. 8.6%)，然而其卻有較高的機率需要二次治療(15.9% vs. 2.6%)〔127〕。雖然經尿道攝護腺切開術治療攝護腺肥大可得到不錯之療效，但目前缺乏超過五年之長期追蹤結果，所以還無法論定其長期療效〔128, 129〕。

建議(建議等級: A)

經尿道攝護腺切開術對於年輕病患，性生活活躍或仍想保有生育能力且其攝護腺體積小於 30 毫升之病患是可考慮之替代治療〔130, 47〕。

C. 經尿道攝護腺電燒汽化術 (Transurethral electrovaporization of the prostate)

經尿道攝護腺電燒汽化術是由 Kaplan 和 Te 於西元 1995 年發表的治療〔131〕。其手術方式是將傳統裝於切除內視鏡上之電燒環換成特殊的金屬滾輪，將設定的高能量電流傳導至滾輪上，滾輪於攝護腺上來回滾動，使得攝護腺組織逐漸汽化，至所需之深度爲止，並且將殘餘組織封住避免流血，其所需時間與經尿道攝護腺切除術相當。和傳統經尿道攝護腺切除術比較的結果，經尿道攝護腺電燒汽化術其短時間之療效一樣好；病人不論是症狀、尿流速或生活品質，皆獲得改善〔132, 133〕。然而術後刺激性排尿症狀、排尿困難及尿滯留，以及非預期性的導尿比例卻較高；而且其選擇之病患的攝護腺體積約爲 35 毫升，故而對於較大體積之攝護腺治療效果，目前並無結果。所以經尿道攝護腺電燒汽化術是否能真正優於經尿道攝護腺切除術，還需要長時間的比較試驗才能知道。

建議 **(建議等級: B)**

經尿道攝護腺電燒汽化術因有較高之手術安全性，故而可考慮應用於手術風險性較高且中度良性攝護腺肥大之病患。

D. 敞開式攝護腺切除術

敞開式攝護腺切除術是指經由恥骨上、恥骨後或會陰部切除攝護腺。經恥骨上及會陰部術式進行的併發症較多，較少施行；目前多採用經恥骨後術式。病患攝護腺體積大於 80 至 100 毫升以上；同時合併有較大膀胱結石或需同時切除膀胱憩室時則建議施行敞開式攝護腺切除術〔134 ~137〕。

結論: **(建議等級: A)**

攝護腺切除術，包括(敞開式，經尿道攝護腺切除術，經尿道攝護腺切開術，經尿道攝護腺汽化術)的結果在主觀和客觀的改善均優於藥物或其他低侵犯性的治療。

E. 雷射手術治療

依據雷射的能量不同，雷射治療可分成三大類：

(1) 經尿道雷射凝固術（Transurethral laser coagulation）**(建議等級 C)**

使用低能量雷射將組織凝固而後會壞死、脫落，達到移除阻塞尿道之攝護腺組織的目的。病患術後在國際攝護腺症狀評分、生活品質（quality of life）及尿流速的改進上都與經尿道攝護腺切除術相當，但術後尿管放置時間較長，尿管重置率亦較高約21%，(經尿道攝護腺切除術約 5%) ，術後刺激性排尿症狀 (66%)比攝護腺切除術後（15%）要高出許多〔47〕。

(2) 經尿道雷射汽化術（Transurethral laser vaporization）**(建議等級 B)**

利用較高的能量將雷射光纖直接接觸在攝護腺上，使用時雷射原與組織保持在近乎接觸的狀態下，予以汽化。以達到類似經尿道攝護腺切除術造成之空洞（TUR-like tunnel）。依目前較大規模之統計〔138, 139〕，術中出血量少，極少有病患因手術而需輸血（0-1%），其術後造成之併發症，如排尿困難（6-9.6%）、膀胱頸攣縮（1.4-2%）、暫時性尿滯留（1-5%）。適用於服用抗凝血劑及有心肺功能疾患之高危險群病患身上〔140〕。術中使用生理食鹽水爲沖洗液，故少有水中毒的危險。但仍需大規模與長期的追蹤與統計以資佐證。

(3) 經尿道雷射切除術（Transurethral laser resection）**(建議等級 B)**

以雷射爲能量來源，進行攝護腺切除手術，但須搭配特殊設計的膀胱鏡，此種手術方式在目前統計上，其手術後國際攝護腺症狀評分、生活品質、尿流速的改善上，與經尿道攝護腺切除術相當，其所造成術後併發症，以目前常用的釹雷射爲例，較大規模的統計如尿管重置率（2.7% ）、尿路感染（2.7% ）、尿失禁（1.1% ）、尿道狹窄（1.9% ）、膀胱頸攣縮（1.5% ），均與傳統經攝護腺切除術相當〔141〕。在出血量及輸血比例上則有減少。但仍需大規模的追蹤與統計以資佐證。經尿道雷射攝護腺切除術已可應用於治療較大(>100 公克)的攝護腺上，在有經驗醫師執刀下，統計結果目前與敞開式攝護腺切除術無異〔142〕。不過此項手術至今仍無術後兩年以上的結果發表，其缺點爲需較長的手術訓練時間，與需特殊器械以進行此項手術。

另類治療

草本植物治療製劑(phytotherapeutic agents)，是用草本植物中提煉萃取某些活性成分物質來治療特定性疾病。此種治療廣爲一般民眾接受，在美國雖然在食品添加物及健康教育法案中，明令草本製劑的製造商只能宣稱改變體能促進健康而不能聲明對某種特定疾病有預防或治療的效果〔143〕，但這類製劑的使用量仍高。在美國，因爲下泌尿道症狀及良性攝護腺肥大原因求診泌尿專科醫師的病人中，就有 30%至 90%正服用草本製劑，在歐洲大陸更是經常使用〔144,145〕。

目前使用在治療下泌尿道症狀及良性攝護腺肥大的草本植物製劑列表於表 3 中〔144, 146〕，這些製劑有些單獨使用有些則合併使用。這些草本植物的萃取物含有多種成分物質，包括 phytosterols(β-sitosterol、campesterol、stigmasterol) 、lupenone、lupeol、terpenoids、battyaids、fectin、plantoil、polysaccharides、blavonoids、phytoestrogens、coumestrol、genistein、Δ5-sterols 及 Δ7-sterols〔144〕，由於成分物質多樣性及複雜性，目前仍不知其具有真正藥理治療作用中最重要的成分爲何？或每天要服用多大劑量？且植物生產地也不同，生長期間的環境氣候不同，萃取方法的不同，其所含成分物質比例也有相當差異，這對於治療良性攝護腺肥大機轉的研究有相當困難度。截至目前爲止尚有許多偏方與治療並無法由證據醫學而得到驗證，因此只將於醫學文獻搜尋得到且經過臨床試驗證實者，才列入討論範圍。

1. Saw Palmetto (Permixon®)

Saw palmetto 是目前治療良性攝護腺肥大及下泌尿道症狀最常使用的草本植物製劑。目前認爲最可能藥理作用在於 5-α 還原酶的抑制，但並未得到共識。在近年來臨床的的實驗結果，Saw palmetto(Permixon®)並未具有特定指標的效果，如在和安慰劑比較實驗中，國際攝護腺症狀評分、最大尿流速、國際勃起功能評估上均無明顯差異〔147, 148〕。和其他 5α 還原酶抑制劑及甲型腎上腺素阻斷劑比較，亦無有效的結論〔149~153〕。

2. South African Star Grass

這類草本製劑中 β-sitosterol 的成分被認爲是主要活性物質，有人認爲其有抗發炎作用或影響攝護腺中 transforming growth factor-β，但和臨床治療上關係不明。雖然有些臨床的實驗，但結果仍有可議，需長期效果安全上的觀察〔154~157〕。

3. Rye-pollen extract (Cernilton®) (Secale cereale)

源於瑞典南部某種植物的花粉，商品名爲 Cernilton®。Cernilton®屬於從裸麥草花粉(rye grass pollen)所萃取出來的物質，含有大量的植物固醇成份(phytosterols)。於治療下泌尿道症候的機轉不明。曾一篇系統性回傾 4 個臨床試驗，顯示 Cernilton® 確有助下泌尿道症候，但試驗均人數太少，時間太短，且藥物品質不明〔158〕。

4. Pygeum Africanum

Pygeum Africanum 屬於非洲李屬植物樹皮所萃取出來的物質，於歐洲及美洲被廣汎地使用於良性攝護腺肥大的治療。其真正的作用機轉並不清楚，不過可能與調整膀胱逼尿肌收縮、抗發炎作用、抑制 fibroblast 增生或是直接增加攝護腺液分泌有關。雖然有許多臨床試驗證實 Pygeum Africanum 可以改善因良性攝護腺肥大引起症狀的緩解，而且口服用時耐受性佳，並沒有特殊的副作用報告，不過絕大部份的試驗缺乏安慰劑組的對照及長期的追蹤〔143~ 145〕。

5. Pumpkin seed

雖然在台灣以 Pumpkin seed 用來改善或預防良性攝護腺肥大及其產生的症狀，是一種普遍的方式，但是於文獻的搜尋上卻發現屬於 Pumpkin seed 治療良性攝護腺肥大之隨機、雙盲及安慰劑組對照的人體臨床試驗，卻是非常的少。不過在動物實驗的研究上有證據顯示 Pumpkin seed 可以抑制大白鼠因睪固酮引起增生的攝護腺〔146, 147〕。Pumpkin seed 含有 β-sitosterol 及其他植物固醇成份(phytosterols)，其作用機轉不明，但可能與膽固醇代謝及抗氧化、抗發炎作用相關〔148, 149〕。

■結論

草本植物製劑用於治療良性攝護腺肥大及下泌尿道症狀已廣爲大眾使用，但目前對這些製劑仍有許多疑問，更待持續廣泛的臨床研究才能解答。

這些疑問包括:

1.對於治療良性攝護腺肥大效果不明

2.對良性攝護腺肥大作用機轉不明

3.主要的作用活性成分不明

4.製劑本身存在很大的差異性

5.安全性未被證實。

建議（建議等級：B）

因上述原因，所以草本植物製劑在治療良性攝護腺肥大及下泌尿道症狀，目前並不建議爲標準治療法。

表 3

ORIGIN OF EXTRACTS USED TO TREAT BPH/LUTS

Scientific Name	Common Name	Branded Product
Serenoa repens	Saw palmetto berry	Permixon®
Sabal serrulata	American dwarf Palm	
Pygeum africanum	African plum tree	Tadenan®
Secale cereale	Rye pollen	Cerniton®
Hypoxis rooperi	South African star grass	Harzol®
Urtica dioica	Stinging nettle	Bazoton®
Cucurbita pepo	Pumpkin seeds	

證據等級列表

Reference Number	Article Title	Evidence Level
Chulte CG. 1993(1)	The prevalence of prostatism: a population based survey of urinary symptoms.	2++
Arrighi HM. 1991 (3)	Natural history of benign prostatic hyperplasia and risk of prostatectomy, the Baltimore Longitudinal Study of Aging.	2++
Wolfs GGMC. 1994 (4)	Prevalence and detectionof micturition problems among 2,734 elderly men.	1+
Tsukamoto T. 1995 (5)	Prevalence of prostatism in Japanese men in a population based study with comparison to a similar American study.	2++
Masumori N. 1996 (6)	Japanese men have smaller prostate volumes but comparable urinary flow rates relative to American men: results of community based studies in 2 countries.	2+
Anderson JB. 2001 (7)	The progression of benign prostatic hyperplasia: examining the evidence and determining the risk.	1+
Jacobsen SJ. 1996 (8)	Natural history of prostatism: longitudinal changes in voiding symptoms in community dwelling men.	2++
McConnell JD. 1998 (9)	The effect of finasteride on the risk of acute urinary retention and the need for surgical treatment among men with benign prostatic hyperplasia.	1++
Meigs JB. 1999 (10)	Incidence rates and risk factors for acute urinary retention: the Health Professional Follow up Study.	1++
Garraway WM, Alexander FE. (11)	Prostate disease: epidemiology, natural history and demographic shifts.	2+
Ranjan P, Dalela D, Sankhwar SN(12)	Diet and benign prostatic hyperplasia: implications for prevention.	2+

Wei JT, Schottenfeld D, Cooper K, et al(13)	The natural history of lower urinary tract symptoms in black American men: relationships with aging, prostate size, flow rate and bothersomeness.	2+
Araki H, Watanabe H, Mishina T, Nakao M(14)	High-risk group for benign prostatic hypertrophy.	2+
Glynn RJ, Campion EW, Bouchard GR, Silbert JE(15)	The development of benign prostatic hyperplasia among volunteers in the Normative Aging Study.	2++
Moon TD, Brannan W, Stone NN, et al(16)	Effect of age, educational status, ethnicity and geographic location on prostate symptom scores.	2+
Padley R, Olson P, Oesterling JE, et al(17)	Education and socio-economic status influence patterns of response to therapy with the alpha adrenergic receptor blocker terazosin and placebo in men with clinical benign prostatic hyperplasia (BPH)	2+
Badia X, Rodriguez F, Carballido J, et al(18)	Influence of sociodemographic and health status variables on the American Urological Association symptom scores in patients with lower urinary tract symptoms.	2+
Ekman P(19)	BPH epidemiology and risk factors.	2+
Sweeney M, Roehrborn C, Boyle P, et al(20)	Sexual dysfunction in European patients with benign prostatic hyperplasia.	2+
Rosen R, Altwein J, Boyle P, et al(21)	Lower urinary tract symptoms and male sexual dysfunction: the multinational survey of the aging male (MSAM-7).	2++
Jakobsen H, Torp-Pedersen S, Juul N(22)	Ultrasonic evaluation of age-related human prostatic growth and development of benign prostatic hyperplasia.	2-
Chopra IJ, Tulchinsky D, Greenway FL(23)	Estrogen-androgen imbalance in hepatic cirrhosis. Studies in 13 male patients.	2-
Platz EA, Kawachi I, Rimm EB, Willett WC,	Race, ethnicity and benign prostatic hyperplasia in the health professionals	2++

Giovannucci E(24)	follow-up study.	
Kang D, Andriole GL, Van De Vooren RC, et al(25)	Risk behaviours and benign prostatic hyperplasia.	2+
Haidinger G, Temml C, Schatzl G, et al(26)	Risk factors for lower urinary tract symptoms in elderly men.	2+
Joseph MA, Harlow SD, Wei JT, et al(27)	Risk factors for lower urinary tract symptoms in a population-based sample of African-American men.	2++
Robson MC(28)	Cirrhosis and prostatic neoplasms.	2+
Cetinkaya M, Cetinkaya H, Ulusoy E, et al(29)	Effect of postnecrotic and alcoholic hepatic cirrhosis on development of benign prostatic hyperplasia.	2+
Boyle P, Napalkov P(30)	The epidemiology of benign prostatic hyperplasia and observations on concomitant hypertension.	2++
Sidney S, Quesenberry C Jr, Sadler MC, et al(31)	Risk factors for surgically treated benign prostatic hyperplasia in a prepaid health care plan.	2++
Daniell HW(32)	Larger prostatic adenomas in obese men with no associated increase in obstructive uropathy.	2+
Seitter WR, Barrett-Connor E(33)	Cigarette smoking, obesity, and benign prostatic hypertrophy: A prospective population-based study.	2+
Chyou PH, Nomura AM, Stemmermann GN, Hankin JH(34)	A prospective study of alcohol, diet, and other lifestyle factors in relation to obstructive uropathy.	2+
Lagiou P, Wuu J, Trichopoulou A, et al(35)	Diet and benign prostatic hyperplasia: a study in Greece.	2+
Giovanucci E, Rimm EB, Chute CG, et al(36)	Obesity and benign prostatic hyperplasia.	2+
Soygur T, Kupeli B,	Effect of obesity on prostatic hyperplasia: Its	2+

Aydos K, et al(37)	relation to sex steroid levels.	
Hammarsten J, Hogstedt B(38)	Clinical, anthropometric, metabolic and insulin profile of men with fast annual growth rates of benign prostatic hyperplasia.	2+
Roehrborn CG, 1997 (39)	Correlation between prostate size estimated by digital rectal examination and measured by transrectal ultrasound.	1+
Roehrborn CG, 2001 (40)	Interexaminer reliability and validity of a three-dimensional model to assess prostate volume by digital rectal examination.	1+
Stamey TA, 1987 (41)	Prostate specific antigen as a serum marker for adenocarcinoma of the prostate.	1+
Roehrborn CG, 1999 (42)	Serum prostate specific antigen as a predictor of prostate volume in men with benign prostatic hyperplasia.	1+
Vesely S, 2003 (43)	Relationship between age, prostate volume, prostate-specific antigen, symptom score and uroflowmetry in men with lower urinary tract symptoms.	1+
Roehrborn CG, 1999 (44)	Serum prostate-specific antigen and prostate volume predict long-term changes in symptoms and flow rate: results of a four-year, randomized trial comparing finasteride versus placebo.	1++
Roehrborn CG, 2001 (45)	Clinical predictors of spontaneous acute urinary retention in men with LUTS and clinical BPH: a comprehensive analysis of the pooled placebo groups of several large clinical trials.	1++
Roehrborn CG, 2000 (46)	Serum prostate specific antigen is a strong predictor of future prostate growth in men with benign prostatic hyperplasia.	1++
AUA Guideline, 2003 (47)	AUA guideline on management of benign prostatic hyperplasia (2003). Chapter 1:	1++

	Diagnosis and treatment recommendations	
Chatelain C, 2001 (48)	Proceedings of the Fifth International Consultation on BPH	5
Griffiths D, 1997 (49)	Standardization of terminology of lower urinary tract function: pressure-flow studies of voiding, urethral resistance and urethral obstruction. International Continence Society Subcommittee on Standardization of Terminology of Pressure-Flow Studies.	1+
Gisolf KWH, 2000 (50)	Analysis and reliability of data from 24-hour frequency-volume charts in men with lower urinary tract symptoms due to benign prostatic hyperplasia.	1+
Van Venrooij GEPM, 2001 (51)	Data from frequency-volume charts versus symptom scores and quality of life score in men with lower urinary tract symptoms due to benign prostatic hyperplasia.	2++
Blanker MH, 2000 (52)	Normal voiding patterns and determinants of increased diurnal and nocturnal voiding frequency in elderly men.	1+
Matthiesen TB, 1999 (53)	Nocturia and polyuria in men referred with lower urinary tract symptoms, assessed using a 7-day frequency-volume chart.	2+
Djavan B. 1999 (58)	Meta-analysis on the efficacy and tolerability of alpha1-adrenoceptor antagonists in patients with lower urinary tract symptoms suggestive of benign prostatic obstruction.	1++
Barry MJ. 1995 (59)	Benign prostatic hyperplasia specific health status measures in clinical research: how much change in the American Urological Association symptom index and the benign prostatic hyperplasia impact index is perceptible to patients?	2+
Andersen JT. 1995 (61)	Can finasteride reverse the progress of benign	1+

	prostatic hyperplasia? A two year placebo-controlled study. The Scandinavian BPH study group.	
Gormley GJ. 1992 (62)	The effect of finasteride in men with benign prostatic hyperplasia.	1+
Nickel JC. 1996 (63)	Efficacy and safety of finasteride therapy for benign prostatic hyperplasia: results of a 2-year randomised controlled trial (the PROSPECT Study).	1+
Vaughan D. 2002 (64)	Long-term (7 to 8-year) experience with finasteride in men with benign prostatic hyperplasia.	2+
Boyle P. 1996 (65)	Prostate volume predicts the outcome of treatment of benign prostatic hyperplasia with finasteride: meta-analysis of randomized clinical trials.	1++
Andersen JT. 1997 (66)	Finasteride significantly reduces acute urinary retention and need for surgery in patients with sympomatic benign prostatic hyperplasia.	1+
McConnell JD. 1998 (67)	The effect of finasteride on the risk of acute urinary retention and the need for surgical treatment among men with benign prostatic hyperplasia. Finasteride Long-term Efficacy and Safety Study Group.	1++
Roehrborn CG. 2000 (68)	Urinary retention in patients with BPH treated with finasteride or placebo over 4 years. Characterization of patients and ultimate outcomes. The PLESS Study Group.	1++
Hudson PB. 1999 (70)	Efficacy of finasteride is maintained in patients with benign prostatic hyperplasia treated for 5 years. The North American Finasteride Study Group.	1+
Ekman P. 1998 (71)	Maximum efficacy of finasteride is obtained	2++

	within 6 months and maintained over 6 years. Follow-up of the Scandinavian Open-extension Study. The Scandinavian Finasteride Study Group.	
Lam JS. 2003 (72)	Long-term treatment with finasteride in men with symptomatic benign prostatic hyperplasia: 10-year follow-up.	2++
Roehrborn CG. 2002 (75)	On behalf of the ARIA3001, ARIA3002 and ARIA3003 study investigators. Efficacy and safety of a dual inhibitor or 5-alphareductase types 1 and 2 (dutasteride) in men with benign prostatic hyperplasia.	1+
Andriole GL. 2003 (76)	Safety and tolerability of the Dual 5 alpha-Reductase Inhibitor dutasteride in the treatment of benign prostatic hyperplasia.	1++
Foley SJ. 2000 (77)	A prospective study of the natural history of hematuria associated with benign prostatic hyperplasia and the effect of finasteride.	1-
Kearney MC. 2002 (78)	Clinical predictors in the use of finasteride for control of gross hematuria due to benign prostatic hyperplasia.	2-
Wessells H. 2003 (79)	PLESS Study Group. Incidence and severity of sexual adverse experiences in finasterides and placebo-treated men with benign prostatic hyperplasia.	1-
Matsumoto AM. 2002 (80)	The long-term effect of specific type II 5alpha-reductase inhibition with finasteride on bone mineral density in men: results of a 4-year placebo controlled trial.	1+
Oesterling JE. 1997 (81)	Biologic variability of prostate specific antigen and its usefulness as a marker for prostate cancer: effects of finasteride. The Finasteride PSA Study Group.	1+
Andriole GL. 1998 (82)	Treatment with finasteride preserves	1++

	usefulness of prostate specific antigen in the detection of prostate cancer: results of a randomized, double-blind, placebo-controlled clinical trial. PLESS Study Group. Proscar Long-term Efficacy and Safety Study.	
Yang XJ. 1999 (83)	Does long-term finasteride therapy affect the histologic features of benign prostatic tissue and prostate cancer on needle biopsy? PLESS Study Group. Proscar Long-term Efficacy and Safety Study.	1-
Keetch DW. 1997 (84)	Comparison of percent free prostate specific antigen levels in men with benign prostatic hyperplasia treated with finasteride, terazosin or watchful waiting.	1-
Pannek J. 1998 (85)	Influence of finasteride on free and total serum prostate specific antigen levels in men with benign prostatic hyperplasia.	1-
McConnell JD. 2003 (86)	The long-term effect of doxazosin, finasteride, and combination therapy on the clinical progression of benign prostatic hyperplasia.	1++
Baldwin KC. 2001 (87)	Discontinuation of alpha-blockade after initial treatment with finasteride and doxazosin in men with lower urinary tract symptoms and clinical evidence of benign prostatic hyperplasia.	1+
Barkin J. 2003 (88)	Alpha-blocker therapy can be withdrawn in the majority of men following initial combination therapy with the dual 5 alpha reductase inhibitor dutasteride.	1+
Reynard JM. 2004(89)	Does anticholinergic medication have a role for men with lower urinary tract symptoms/benign prostatic hyperplasia either alone or in combination with other	2++

	agents?	
Blake-James BT. 2007 (91)	The role of anticholinergics in men with lower urinary tract symptoms suggestive of benign prostatic hyperplasia: a systematic review and meta-analysis.	2+
Abrams P. 2006 (92)	Safety and tolerability of tolterodine for the treatment of overactive bladder in men with bladder outlet obstruction.	2+
Lee KS. 2005 (93)	Combination treatment with propiverine hydrochloride plus doxazosin controlled release gastrointestinal therapeutic system formulation for overactive bladder and coexisting benign prostatic obstruction: a prospective, randomized, controlled multicenter study.	1+
Athanasopoulos A. 2003 (94)	Combination treatment with an alpha-blocker plus an anticholinergic for bladder outlet obstruction: a prospective, randomized, controlled study.	1+
Ruggieri MR Sr. 2005 (95)	Combined use of alpha-adrenergic and muscarinic antagonists for the treatment of voiding dysfunction.	2++
Foster, RS. 1993 (96)	High-intensity focused ultrasound in the treatment of prostatic disease	2-
Schulman CC. 1993 (98)	Hyperthermia and thermotherapy of benign prostatic hyperplasia: a critical review.	2-
Matzkin H. 1994 (99)	Hyperthermia as a treatment modality in benign prostatic hyperplasia.	3
Abbou CC. 1995 (100)	Transrectal and transurethral hyperthermia versus sham treatment in benign prostatic hyperplasia: a double-blind randomized multicentre clinical trial. The French BPH Hyperthermia.	2++
De la Rosette JJ. 1997	Clinical results of strategies to reduce	4

(101)	morbidity in high energy transurethral microwave thermotherapy (HE-TUMT).	
Rodrigues Netto N. 1994 (102)	Ejaculatory dysfunction after transurethral microwave thermotherapy for treatment of benign prostatic hyperplasia.	2-
Francisca EA. 1997 (103)	Quality of life assessment in patients treated with lower energy thermotherapy (Prostasoft 2.0): results of a randomized transurethral microwave thermotherapy versus sham study.	2+
D'Ancona FC. 1997 (104)	High energy thermotherapy versus transurethral resection in the treatment of benign prostatic hyperplasia (BPH): results of a prospective randomized study with 1-year follow-up.	2+
Chapple CR. 1999 (105)	Transurethral needle ablation (TUNA). A critical review of radiofrequency thermal therapy in the management of benign prostatic hyperplasia.	2+
Naslund, MJ. 1997 (106)	Transurethral needle ablation of the prostate.	2+
Bruskewitz R. 1998 (107)	A prospective randomized 1-year clinical trial comparing transurethral needle ablation to transurethral resection of the prostate for the treatment of symptomatic benign prostatic hyperplasia.	1+
Ramon J. 1997 (108)	Transurethral needle ablation of the prostate for the treatment of benign prostatic hyperplasia: a collaborative multicentre study.	2+
Schulman CC. 1997 (109)	Transurethral needle ablation (TUNA) of the prostate: clinical experience with two years' follow-up in patients with benign prostatic hyperplasia (BPH).	2-
Schatzl G. 1997 (110)	The early postoperative morbidity of	2-

	transurethral resection of the prostate and of four minimally invasive treatment alternatives.	
Zlotta AR. 2003 (111)	Long-term evaluation of transurethral needle ablation of the prostate (TUNA) for treatment of symptomatic benign prostatic hyperplasia: clinical outcome up to five years from three centers.	2+
Rosario DJ. 1997 (112)	Safety and efficacy of transurethral needle ablation of the prostate for symptomatic outlet obstruction.	2++
Millard RJ. 1996 (113)	A study of the efficacy and safety of transurethral needle ablation (TUNA®) treatment for benign prostatic hyperplasia.	2-
Steele GS. 1997 (1014)	Transurethral needle ablation of the prostate: a urodynamic based study with 2-year follow-up.	2+
Schulman CC. 1998 (115)	Transurethral needle ablation (TUNA™) of the prostate: clinical experience with three years follow-up in patients with benign prostatic hyperplasia (BPH).	2+
Madersbacher S. 1996 (116)	High-intensity focused ultrasound for prostatic tissue ablation.	2-
Madersbacher S. 1996 (117)	The urodynamic impact of transrectal high intensity focused ultrasound on bladder outflow obstruction.	2+
McConnell J.D., 1994(118)	Clinical Practice Guideline for Benign Prostatic Hyperplasia: Diagnosis and Treatment, Agency for Health Care Policy and Research, Rockville, Maryland	1++
Wasson J.H., 1995(119)	A comparison of transurethral surgery with watchful waiting for moderate symptoms of benign prostatic hyperplasia. The Veterans Affairs Cooperative Study Group on	1++

	Transurethral Resection of the Prostate	
Mebust WK, 1989 (120)	Transurethral prostatectomy: immediate and postoperative complications – cooperative study of 13 participating institutions evaluating 3885 patients	1+
Borboroglu PG 1999(121)	Immediate and postoperative complications of transurethral prostatectomy in 1990s	1+
Jens Rassweiler 2006(122)	Complications of Transurethral Resection of the Prostate (TURP)—Incidence, Management, and Prevention	1+
Orandi A. 1985(123)	Transurethral incision of prostate (TUIP): 646 cases in 15 years—a chronological appraisal	2++
Riehmann M. 1995(124)	Transurethral resection versus incision of the prostate: a randomized, prospective study	1+
Saporta L. 1996(125)	Objective and subjective comparison of transurethral resection, transurethral incision and balloon dilatation of the prostate	2++
Sparwasser C. 1995(126)	Long-term results of transurethral prostate incision (TUIP) and transurethral prostate resection (TURP). A prospective randomized study	1+
Madersbacher, 1999(127)	Is transurethral resection of the prostate still justified?	2+
Yang Q. 2001(128)	Transurethral incision compared with transurethral resection of the prostate for bladder outlet obstruction: a systematic review and meta-analysis of randomized controlled trials	1+
Tkocz M., 2002(129)	Comparison of long-term results of transurethral incision of the prostate with transurethral resection of the prostate, in patients with benign prostatic hypertrophy	2++
Madersbacher S., 2004(130)	EAU 2004 guidelines on assessment, therapy and follow-up of men with lower urinary	1++

	tract symptoms suggestive of benign prostatic obstruction (BPH guidelines)	
Kaplan S.A. 1995(131)	Transurethral electrovaporization of the prostate: a novel method for treating men with benign prostatic hyperplasia	2++
Hammadeh M.Y. 2003(132)	5-year outcome of a prospective randomized trial to compare transurethral electrovaporization of the prostate and standard transurethral resection	1+
H.H. van Melick, 2003(133)	Long-term follow-up after transurethral resection of the prostate, contact laser prostatectomy, and electrovaporization	1+
Roehrborn, C, 2002(134)	Etiology, pathophysiology, epidemiology and natural history of benign prostatic hyperplasia. In: Campbell's Urology, 8th ed	1++
Tubaro A, 2001(135)	A prospective study of the safety and efficacy of suprapubic transvesical prostatectomy in patients with benign prostatic hyperplasia.	1+
Mearini E, 1998(136)	Open prostatectomy in benign prostatic hyperplasia: 10-year experience in Italy	1-
Serretta V. 2002(137)	Open prostatectomy for benign prostatic enlargement in southern Europe in the late 1990s: a contemporary series of 1800 interventions	1+
Malek. 2005 (138)	Photoselective Potassium-Titanyl-Phosphate laser vaporization of the benign obstructive prostate	2+
Te AE. 2006 (139)	Impact of prostate-specific antigen level and prostate volume as predictors of efficacy in photoselective vaporization prostatectomy: analysis and results of an ongoing prospective multicentre study at 3 years.	2+
Reich. 2005 (140)	High power (80 W) potassium-titanyl –phosphate laser vaporization of the	2+

	prostate in 66 high risk patients	
Elzayat. 2005 (141)	Holmium laser enucleation of the prostate: A size-independent new “gold standard”	2++
Moody, J A. 2005 (142)	Holmium laser enucleation for prostate adenoma greater than 100 gm	2-
Willetts KE. 2003(147)	Serenoa repens extract for benign prostate hyperplasia:a randomized controlled trial.	1+
Carraro JC.1996 (149)	Comparison of phytotherapy (Permixon) with finasteride in the treatment of benign prostate hyperplasia: a randomized international study of 1,098 patients.	1+
Debruyne F. 2002 (150)	Comparison of a phytotherapeutic agent (Permixon) with an alpha-blocker(tamsulosin) in the treatment of benign prostatic hyperplasia: a 1-year randomized international study.	1+
Debruyne F. 2004 (151)	Evaluation of the clinical benefit of permixon and tamsulosin in severe BPH patients – PERMAL study subset analysis.	1+
Zlotta AR. 2005(152)	Evaluation of male sexual function in patient with lower urinary tract symptoms (LUTS) associated with benign prostatic hyperplasia(BPH) treated with a phytotherapeutic agent (Permixon), tamsulosin or finasteride.	1+
Glemain P. 2004 (152)	Tamsulosine avec ou sans Serenoa repens dans I’hypertrophie benigne de la prostate	1
Berges RR. 1995 (154)	Randomised, placebocontrolled, double-prostatic hyperplasia.	1
Berges RR. 2000 (155)	Treatment of symptomatic benign prostatic hyperplasia with β-sitosterol: and 18-month follow-up.	2-
Klippel KF. 1997 (156)	A multicentric, placebo-controlled, double-blind clinical trial of beta-sitosterol	1

	(phytos-terol) for the treatment of benign prostatic hyperplasia.	
Wilt TJ. 1999 (157)	β-sitosterol for the treatment of benign prostatic hyperplasia: a systematic review.	1-
MacDonald R. 2000 (158)	A systematic review of Cernilton for the treatment of benign prostatic hyperplasia.	1-

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