

## REVIEW

# Vacuum therapy in erectile dysfunction—science and clinical evidence

J Yuan<sup>1</sup>, AN Hoang<sup>1</sup>, CA Romero<sup>1</sup>, H Lin<sup>2</sup>, Y Dai<sup>2</sup> and R Wang<sup>1</sup>

<sup>1</sup>Division of Urology, University of Texas Medical School at Houston, Houston, TX, USA and <sup>2</sup>Affiliated Drum Tower Hospital, Nanjing University School of Medicine, Nanjing, Jiangsu Province, China

Vacuum therapy (VT) utilizes negative pressure to distend the corporal sinusoids and to increase the blood inflow to the penis. Depending on its purpose, VT could be used as vacuum constriction device (VCD), with the aid of an external constricting ring which is placed at the base of penis to prevent blood outflow, maintaining the erection for sexual intercourse. Also, as a vacuum erectile device (VED), without the application of a constriction ring, just increases blood oxygenation to the corpora cavernosa and for other purposes. The emerging of phosphodiesterase 5 inhibitors (PDE<sub>5</sub>I) for the treatment of erectile dysfunction (ED) eclipsed VCD as therapeutic choice for ED; however, widespread usage of VED as part of penile rehabilitation after radical prostatectomy and other purposes rekindle the interest for VT. The underlying hypothesis is that the artificial induction of erections shortly after surgery facilitates tissue oxygenation, reducing cavernosal fibrosis in the absence of nocturnal erections, and potentially increases the likelihood of preserving erectile function. Due to its ability to draw blood into the penis regardless of nerve disturbance, VED has become the centerpiece of penile rehabilitation protocols. Herein, we reviewed the history, mechanism, application, side effects and future direction of VT in ED.

*International Journal of Impotence Research* (2010) 22, 211–219; doi:10.1038/ijir.2010.4;  
published online 22 April 2010

**Keywords:** vacuum constriction device; vacuum erectile device; erectile dysfunction; penile rehabilitation; radical prostatectomy

## Introduction

Vacuum therapy (VT) uses negative pressure to distend the corporal sinusoids and to increase blood inflow to the penis. If a constricting ring is used, VT would be categorized as a vacuum constriction device (VCD); in this setting, an external constricting ring is placed at the base of the penis to prevent blood outflow from the corpora cavernosa, and an erection is maintained for sexual intercourse. A vacuum erectile device (VED) is the use of VT without a constrictive ring, with the objective of increasing blood oxygenation in the corpora cavernosa, among other reasons. As a non-invasive, effective, safe, drug-free and cost-effective erectile dysfunction (ED) treatment, VCD was gradually

accepted by the urological community and was finally recommended as an alternative for treatment of ED by the American Urological Association in 1996.<sup>1</sup> With the great success of phosphodiesterase-5 inhibitors (PDE<sub>5</sub>I) for treatment of ED, VCD lost its luster. However, when PDE<sub>5</sub>I limitations were shown and VED use expanded, interest about VT has been rekindled. This review addresses the applications of VT for ED, penile rehabilitation (PR) and other urological conditions.

## History

In 1874, John King, an American physician, stated that ‘when there is impotency with a diminution of the size of the male organ, the glass exhauster should be applied to the part’. What he referred to was simply a vacuum device capable of producing an artificial erection. However, it failed to maintain the erection once the glass exhauster was taken off the penis.<sup>2</sup> It was not until 1917, when a patent was granted to Otto Lederer for his ‘surgical device to produce erection with vacuum’, that the concept of a ‘compression’ ring to be used in conjunction with the

Correspondence: Dr R Wang, Division of Urology, University of Texas Health Science Center at Houston, 6431 Fannin St, MSB 6.018, Houston, TX 77030, USA.  
E-mail: Run.Wang@uth.tmc.edu

Received 2 December 2009; revised 15 January 2010; accepted 14 February 2010; published online 22 April 2010

vacuum device was introduced.<sup>3</sup> Since then, several patents had been granted to modifications,<sup>4,5</sup> but the credit for the popularization of VCD is generally given to a Georgian entrepreneur, Geddis D Osbon, who developed his 'youth equivalent device' in the 1960s. It was reported that he personally used the device for more than 20 years without device failure while popularizing and perfecting the device. His effort culminated in the first marketed VCD device, Erecaid,<sup>6</sup> to receive the US Food and Drug Administration (FDA) approval in 1982.<sup>7</sup> Despite this, the device faced strong skepticism among the medical community and patients. Instrumental in overcoming these skepticisms and popularizing the device were the early works of Nadig<sup>8</sup> and Witherington<sup>9</sup> in establishing its efficacy and safety profiles. It was thought to have finally gained acceptance by the medical community with Lue's commentary in the *Journal of Urology*: 'I recommend a vacuum constriction device to all of my patients (except those with coagulation disorders and sickle cell disease) as the initial medical option'.<sup>10</sup> As more evidence emerged, the American Urological Association ultimately recommended VCD as one of three treatment alternatives for organic ED.<sup>1</sup>

#### Devices and mechanisms

Currently, there are over dozens of commercially available VCDs, that is, Timm Medical Technologies (Eden Prairie, MN, USA), Mission Pharmacal (San Antonio, TX, USA), Encore (Louisville, KY, USA), Mentor (Santa Barbara, CA, USA) and Post-T-Vac (Dodge City, KS, USA). All of these devices share the same basic mechanics since its original development. They all comprise three components: a vacuum cylinder, a battery or manually operated vacuum pump and constriction rings of varying sizes (Figure 1).<sup>11</sup> Some of the latest models have a pressure release valve designed to prevent penile injury from excessive negative pressure.<sup>12</sup> It is reported that single-handed devices are more desirable to novice users.<sup>13</sup>

Usage begins with placing the correct constriction ring over the open end of the vacuum cylinder. A copious amount of a water-soluble lubricant is then applied to the base of the penis to create a tight seal once the vacuum cylinder is placed over. Negative pressure (100–225 mm Hg) generated either by hand or a battery-operated pump is then applied to create an artificial erection.<sup>14</sup> Once the desired state of erection is achieved, the constriction ring is displaced onto the base of the penis to maintain the erect state. Variable vacuum cylinders and constriction rings are available to select for those that are most comfortable and effective. The vacuum cylinders could then be removed and the patient may have intimacy. Patients can become proficient with the device within 5 days<sup>15</sup> or four practice sessions.<sup>9</sup> The time required to achieve an adequate penile

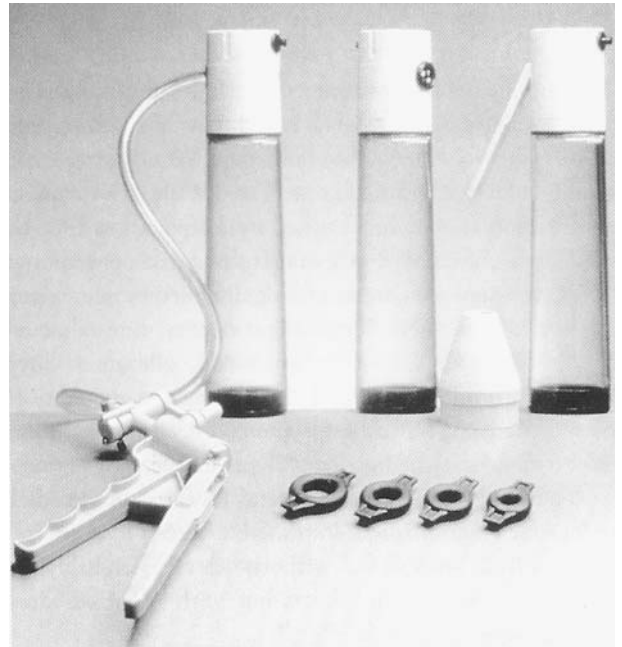


Figure 1 Three types of human vacuum devices.

erection ranges from 30 s to 7 min,<sup>9,16</sup> and many manufacturers advise patients to pump for 1–2 min, release and then pump again for 3–4 min.<sup>7</sup>

Unlike the normal physiology of penile erection in which a complex interplay between neural inputs, vascular patency and hormonal secretion is required, tumescence from VCD resulted from passive flow of mixed venous and arterial blood.<sup>17,18</sup> Broderick *et al.*<sup>17</sup> showed by color Doppler ultrasound that the negative pressure transiently drew arterial blood into the sinusoidal spaces of the cavernosal tissues, increasing its diameter nearly two-fold. The change in diameter owed itself to both intracorporal and extracorporal distention. The constriction ring placed at the base of the penis prevented the venous outflow. Color Doppler ultrasound performed after the placement of constriction ring, however, showed no arterial inflow into the penis.<sup>17</sup> Blood gas analysis showed ischemia after 30 min of applying constriction ring.<sup>18</sup> This led to the recommendation that the constriction ring should not be left on for >30 min to prevent ischemic injury to the penis. Compared with naturally occurring erections, VCD-derived erections are perceived differently by both the man and their partner. The 'erection' looks dusky and feels cooler than normal, with increased volume distally, especially at the glans penis.<sup>7</sup>

#### VT in erectile dysfunction–VCD

##### Indications

VCD therapy can be applied successfully for nearly all etiologies of ED;<sup>8</sup> although its success depends

on appropriate instruction and practice.<sup>19</sup> More than 90% of men will experience functional erection with VCD therapy with adequate practice.<sup>9</sup>

It is currently a second-line therapy along with intracavernosal self-injection and intraurethral therapy with vasoactive substances.<sup>20</sup> It is widely believed that VCD therapy is more acceptable among elderly patients with occasional sexual intimacy, as younger patients may show limited acceptance because of its perceived 'unnatural' erection.<sup>21</sup> Chen *et al.*, conversely, reported that VCD therapy remained the preferred treatment option among couples who had achieved satisfactory erections with either VCDs and PDE<sub>5</sub>I.<sup>22</sup>

VCD may also be used in conjunction with other therapies for synergistic effects. It has been reported that VCD therapy could be combined with PDE<sub>5</sub>I,<sup>23</sup> intracavernosal self-injection,<sup>24,25</sup> intraurethral therapy,<sup>26</sup> psychotherapy<sup>27</sup> and even penile prosthesis.<sup>28,29</sup>

### Contraindications

Contraindications to the use of VCD are few and primarily include patients with a tendency for spontaneous priapism or intermittent prolonged erections, and those with severe penile anomalies (either congenital or acquired).<sup>7</sup> There are some relative contraindications, which can be overcome by education and precautions care, such as cultural taboo, cervical or high-thoracic spinal cord injuries, neurological disease or degenerative joint diseases with poor manual dexterity.<sup>30</sup> Patients with bleeding disorders or those on anticoagulation therapy are considered at high risk to develop petechiae, ecchymosis or hematoma;<sup>7</sup> however, it was shown that the risk did not exceed that of the general population.<sup>31</sup>

### Clinical results

VCDs, unlike PDE<sub>5</sub>Is, have not had to undergo strict clinical trials to show their safety and efficacy before

their widespread usage. A review of literature consists largely of single-center observational series, collection of small prospective clinical trials and commercial databases. Despite the paucity of data, a wealth of clinical experience is available, although with big variability in the clinical efficacy of VCD therapy. These data are present in Table 1.

Nadig *et al.* were the first to provide objective data regarding penile rigidity obtained using VCDs. It was found that buckle pressures of 454 g (the minimal criterion for rigidity used by many sleep laboratories) were achieved in 27/35 (77%) patients.<sup>8</sup> Bosshardt and co-workers showed that a nocturnal penile tumescence rigidity of 80% (70% being sufficient for intercourse)<sup>40</sup> was the norm after 6 months in their group of 26 patients. They also showed that induced ischemia could result after 30 min of applying the constriction rings. This led to the recommendation that the constriction ring should not be left on for >30 min to prevent ischemic injury to the penis.<sup>18</sup>

The effectiveness of VED has been established for different causes of ED. For those with arteriogenic ED, VCD therapy achieved the efficacy and satisfaction rates of 67 and 84%, respectively.<sup>37</sup> For the corporeal veno-occlusive dysfunction ED, Kolettis *et al.*<sup>38</sup> reported a 56% satisfaction rate. For diabetic ED, Arauz-Pacheco *et al.*<sup>41</sup> and Bodansky *et al.*<sup>42</sup> reported successful rates of 75 and 58%, respectively. No severe side effects were reported. For spinal cord injury-induced ED, an impressive 85% successful rate was achieved with minor side effects.<sup>43</sup> The largest patient group (34 777 cases) was reported by Lewis and Witherington<sup>7</sup> who used the VCD data from Osbon data bank with satisfaction rates 65–83%; however, only 17% (5847) cases had enough information to evaluate. Conversely, Vrijhof *et al.*<sup>36</sup> and Sidi *et al.*<sup>16</sup> reported much lower satisfaction rates, 50 and 68%, respectively. More dramatically, Dutta *et al.* reported only 35% satisfaction rate, with an attrition rate of 65%.<sup>39</sup> It is

**Table 1** Efficacy of VCDs

Authors	Year published	Study design	Enrollee (mean follow-up)	Results
Nadig <i>et al.</i> <sup>8</sup>	1986	Prospective	35 (8–22 months)	> 90% achieved adequate erections. 80% use regularly
Witherington <sup>9</sup>	1989	Retrospective	1517 (8.6 months)	92% good erection
Sidi <i>et al.</i> <sup>16</sup>	1990	Prospective	100 (7.9 months)	68% satisfaction rate
Cookson <i>et al.</i> <sup>32</sup>	1993	Retrospective	216 (29 months)	70% use regularly. Quality of erection plus satisfaction > 90%
Segenreich <i>et al.</i> <sup>33</sup>	1993	Prospective	150 (25 months)	75% achieved adequate erection. > 90% satisfaction rate
Blackard <i>et al.</i> <sup>34</sup>	1993	Prospective	45 (?)	69% satisfaction rate
Meinhart <i>et al.</i> <sup>35</sup>	1993	Prospective	74 (3 weeks)	27% satisfaction rate
Vrijhof <i>et al.</i> <sup>36</sup>	1994	Prospective	67	50% achieved adequate erection
Baltaci <i>et al.</i> <sup>37</sup>	1995	Retrospective	61 (12.8 months)	> 80% satisfaction rate. 67% effectiveness rate
Bosshardt <i>et al.</i> <sup>18</sup>	1995	Prospective	30 (6 months)	Quality of erection 80%
Kolettis <i>et al.</i> <sup>38</sup>	1995	Prospective	50	56% satisfaction rate. An acceptable treatment mode for CVOD
Lewis <i>et al.</i> <sup>7</sup>	1997	Retrospective	5847 (?)	65–83%
Dutta <i>et al.</i> <sup>39</sup>	1999	Prospective	129 (37 months)	High attrition rate (65%). 35% satisfaction rate

Abbreviations: CVOD, corporeal veno-occlusive dysfunction; VCD, vacuum constriction device.

**Table 2** Complications of VCDs

<i>Most common</i>	<i>Rare</i>
Penile discomfort/pain	Penile hematoma
Penile coldness	Penile hyper-pigmentation
Penile numbness	Penile skin necrosis
Petechia or bruising	Urethral bleeding
Ecchymosis	Peyronie's disease
Inability to ejaculate	Penile gangrene
Pain on ejaculation	Fournier's syndrome

Abbreviation: VCD, vacuum constriction device.

believed that the huge discrepancy of satisfaction rates may be attributed to patient selection and patient education.

### Complications

The use of VCD is usually well tolerated with mostly mild side effects (Table 2). The most common side effects include numbness, pain, penile bruising or petechiae.<sup>9,32,37,44–46</sup> Due to the constriction rings, painful ejaculation or sensation of trapped ejaculate has also been commonly reported.<sup>7,9</sup> Other rare complications such as leg spasms, testicular migration, urethral varicosities/bleeding have been reported.<sup>47</sup> Major complications like Peyronie's disease, penile skin necrosis, penile gangrene and Fournier's syndrome have been reported anecdotally.<sup>47,48–52</sup>

## VT in penile rehabilitation—VED

### Introduction

Prostate cancer is the most common solid-organ cancer among men and one of the leading causes of death.<sup>53</sup> With early detection and radical prostatectomy (RP), the 15-year overall actuarial cancer-specific survival rate has reached 90%.<sup>54,55</sup> Unfortunately, RP is associated with several quality-of-life side effects, mainly urinary incontinence and ED.<sup>56,57</sup> With improvements in technique and the use of robotic-assisted procedures, incontinence rates have reduced to an acceptable level.<sup>58–61</sup> However, the same cannot be said for ED, as the incidence of ED after RP ranges from 10 to 100%.<sup>62</sup> In an attempt to improve patients' quality of life after RP, PR is now widely applied in clinical practice.<sup>55,63–65</sup> Currently, PR methods include the use of PDE<sub>5</sub>I, intracavernosal self-injection/intraurethral suppositories, VED or a combination of different therapy modalities.<sup>55,63–65</sup>

### Pathophysiology

Erectile function (EF) becomes impaired immediately after RP secondary to cavernous nerve damage during surgery, resulting in neuropraxia.<sup>66</sup> Even

with the most meticulous nerve-sparing dissection, some degree of neuropraxia is unavoidable because of the close proximity of the nerves to the prostatic gland. These nerves tend to recover slowly, and it may take as long as 3 years for them to return to a new baseline functional status.<sup>58</sup> Absence or decreased erection and penile size ensue before recovery of the cavernous nerve.<sup>57,67</sup> A reduction in arterial inflow has also been reported due to ligation of the accessory internal pudendal arteries during RP.<sup>68,69</sup> The combination of nerve damage with decreased arterial inflow may cause penile tissue hypoxia leading to apoptosis and collagen deposition, which ultimately results in venous leak, which in turn, has been linked to the pathophysiology of ED after RP.<sup>70–79</sup> As nerve recovery requires time, it is hypothesized that VED may bypass the neuropraxic period by directly dilating the cavernosal arteries, therefore, overcoming hypoxia and preventing apoptosis and fibrosis before the functional recovery of the cavernous nerve.

### Laboratory and clinical evidence

VED, as PR modality, simulates natural erection and allows reoxygenation of the penis.<sup>80</sup> There has been growing evidence to support this. Blair *et al.*<sup>81</sup> documented that sub-atmospheric pressure induces an initial increase in arterial inflow in the forearm of healthy volunteers; Greenfield and Paterson<sup>82</sup> further showed a vasodilation effect on the arteries of the forearm in volunteers exposed to a sub-atmospheric pressure of  $-150$  to  $-200$  mm Hg, which is similar to the pressure used in VED.<sup>14</sup> Diederichs *et al.*<sup>83</sup> noticed that negative pressure induces expansion of penile tissue followed by increase in blood flow in primates; the authors believed that the increased blood flow was arterial inflow. Broderick *et al.*<sup>17</sup> showed that a transient vacuum application increases the peak flow velocity of the cavernous arteries in humans. Donatucci and Lue<sup>84</sup> further determined that chronic VED usage increases cavernous arterial flow in men with mild vasculogenic ED as measured using a duplex ultrasonograph. Bosshardt *et al.* performed blood gas analysis before VED application, immediately after the application of a constriction ring and repeated blood gas analysis 15 and 30 min later with the constriction ring in place in ED patients. This test showed that the mean O<sub>2</sub> saturation of corporal blood immediately after VED-induced erection was 79.2%, which translates as arterial and venous contributions of flow of 58 and 42%, respectively. In addition, after 30 min of continuous application of a constriction ring, blood gas analysis showed ischemia of the penile blood.<sup>18</sup> This is the rationale for the use of VED as PR modality, instead of VCD. Recently, Müller *et al.*<sup>85</sup> applied hyperbaric oxygen therapy on a bilateral cavernous nerve crush rat model and showed that hyperbaric oxygen

**Table 3** Summary table of penile rehabilitation trials

<i>Authors</i>	<i>Year published</i>	<i>Treatment regimen</i>	<i>Study design</i>	<i>Enrollee</i>	<i>Results</i>
Schwartz <i>et al.</i> <sup>89</sup>	2004	QOD PDE <sub>5</sub> I	Prospective	21	No loss of smooth muscle in 50-mg group, gain of smooth muscle in 100-mg group
Bannowsky <i>et al.</i> <sup>90</sup>	2008	Daily PDE <sub>5</sub> I	Prospective, randomized control	41	Treatment group has significantly higher IIEF and higher spontaneous erection rates
Mccullough <i>et al.</i> <sup>91</sup>	2008	Daily PDE <sub>5</sub> I	Prospective, randomized, placebo control	54	Treatment group had higher return of rigidity, higher rate of spontaneous erections
Raina <i>et al.</i> <sup>92</sup>	2006	Daily VED	Prospective, randomized control	109	Improved sexual satisfaction, higher rate of spontaneous erections
Köhler <i>et al.</i> <sup>93</sup>	2007	Daily VED (10 min), immediate versus delayed	Prospective, randomized	28	Delayed use of VED did not affect sexual satisfaction once use began. No statistical significance in penile shrinkage once VED started
Montorsi <i>et al.</i> <sup>94</sup>	1997	ICI 3 times weekly	Prospective, randomized control	30	Higher percentage of treatment group having spontaneous erections
Mulhall <i>et al.</i> <sup>95</sup>	2005	ICI or PDE <sub>5</sub> I to achieve erections 3 × weekly	Prospective, control	132	Treatment group had 2.7 times the rate of spontaneous erections, statistically higher IIEF scores
Nandipati <i>et al.</i> <sup>96</sup>	2006	Daily PDE <sub>5</sub> I and ICI 2–3 times weekly	Prospective	22	Assisted early sexual activity and satisfaction. Addition of PDE <sub>5</sub> I allows lower dose of ICI

Abbreviations: ICI, intracorporal injection; IIEF, International Index of Erectile Function; PDE<sub>5</sub>I, phosphodiesterase inhibitor; VED, vacuum erectile device.

Source: Hinh P and Wang R: Overview of Contemporary Penile Rehabilitation Therapies. *Advances in Urology*, 2008.

therapy improved the preservation of EF. To explore the molecular mechanism of VED, a rat-specific VED, which simulates human VED, was successfully created and applied on a bilateral cavernous nerve crush model by our group.<sup>86</sup> Our preliminary data indicated that daily VED therapy significantly improved the intracavernosal pressure/mean artery pressure ratio, preserved penile length and girth, decreased the level of hypoxia-inducible factor-1 $\alpha$ , transforming growth factor- $\beta$ 1, collagen and apoptosis, and increased the level of eNOS and  $\alpha$ -smooth-muscle actin.<sup>87</sup>

The latest PR trials are summarized in Table 3.<sup>88</sup> Most trials used PDE<sub>5</sub>I to preserve penile sexual health. One limitation of PDE<sub>5</sub>I is its requirement of intact nerves to produce nitric oxide for proper function. There have been theories that PDE<sub>5</sub>I may work through a separate, neuron-independent endothelial cell mechanism; however, it is still unproven.<sup>80</sup> The direct mechanism of VED circumvented this limitation. Given its low complication rate and relatively high compliance rate, along with being the only modality that preserves penile length, VED is an ideal choice as PR after RP<sup>80</sup> or other pelvic injuries.<sup>97,98</sup>

Munding *et al.*<sup>99</sup> documented that up to 48% men had considerable shortening of the stretched penile length (greater than 1.0 cm) at 3 months after RP. Savoie *et al.*<sup>100</sup> found that nearly 20% of men who undergo RP experience a penile length loss greater than 15%. Gontero *et al.*<sup>101</sup> followed 126 men who

had undergone RP and measured penile length before surgery, at the time of catheter removal, and then at 3, 6 and 12 months. They found that the greatest amount of shrinkage occurs in the immediate postoperative period, although shortening continues at a lesser rate throughout the entire study period. They also found that a return of EF, defined as an International Index of Erectile Function (IIEF) of 15, and a nerve-sparing technique during RP, mitigated penile shaft shrinkage. Several studies looking at the efficacy of VED in preserving EF have also examined preserved penile length as a secondary endpoint.

Raina *et al.*<sup>92</sup> randomized 109 post-NSRP patients to early VED daily usage (group-1,  $n = 70$ ) versus no erectogenic aid (group-2,  $n = 35$ ). The participants were followed by the Sexual Health Inventory for Men (SHIM) score. The secondary endpoints included compliance, changes in penile circumference or length, return of natural EF and ability for vaginal intercourse. At the end of the 9-month follow-up, 80% (60/74) of those in group-1 were able to have sexual intercourse using the device, with a satisfaction rate of 55%. Nineteen patients reported return of natural erections and 17 had erections sufficiently firm for vaginal penetration. Conversely, only 37% (13/37) of the patients in group-2 regained natural erections. When evaluating for secondary endpoints, among those who used the device regularly, only 23% (14/60) reported a decrease in penile length and girth, whereas 22/35

(60%) patients in the control group complained of penile shrinkage. This result was confirmed by Dalkin *et al.* who administered VED therapy to 39 men after RP for 90 days after catheter removal.<sup>102</sup> In their study, 97% of compliant men maintained their preoperative stretched penile length; shrinkage was defined as  $\geq 1$  cm. The authors concluded that early usage of VED facilitates early return of spontaneous EF, early resumption of sexual life resulting in spousal satisfaction and preservation of penile length and size.

Other authors have investigated the timing of VED therapy to maximize its benefits. Köhler *et al.*<sup>93</sup> randomized 28 patients undergoing RP to early VED use (group-1,  $n = 17$ ) and delayed VED use (group-2,  $n = 11$ ). Group-1 was instructed to use VED daily, starting at 1 month after RP for two consecutive 5-min periods. Group-2 was instructed to use VED, as many times as desired, starting at 6 months after RP. These men were followed using IIEF scores. At a follow-up of 3 and 6 months, group-1 had a statistically higher IIEF score than group-2. Beyond 6 months, when group-2 began using VED therapy, there was no statistical difference in IIEF scores between both groups. When evaluating for penile shortening after RP, group-1 did not experience any significant penile length reduction; whereas group-2 experienced considerable shrinkage at 3 months (mean loss 1.87 cm) and 6 months of follow-up (mean loss 1.82 cm). However, when group-2 began using VED therapy, the mean penile length loss decreased to 1 cm. The authors concluded that early VED therapy after RP helps to improve early sexual function and preserve penile length.

Solid evidence that VED daily rehabilitation preserves penile length and EF has been scattered; however, a multicenter, randomized study with objective criteria and long-term follow-up is still needed.

## VT under other urological conditions

In addition to its applications for ED and PR, VT has been expanded to other urological conditions. VED has been regarded as an initiator and enhancement of other ED therapies. Bellorofonte *et al.*<sup>103</sup> showed a synergistic action between intracavernosal vasoactive agents and VT. John *et al.*<sup>26</sup> combined VED with intra-urethral prostaglandin E1 to eliminate the need for constriction ring, with 100% successful rate in 19 patients. Cecchi *et al.*<sup>104</sup> evaluated VT with topical minoxidil in 18 patients and found that the combination enhanced the erection quality, reduced the time of device application and avoided 67% (12/18) the need for constrictive rings.

VED has also been used to preserve or restore natural potency. Oakley *et al.*<sup>11</sup> speculated that VED usage may delay the onset of intractable impotence

in high-risk groups, such as those with diabetes and dyslipidemia. Colombo *et al.*<sup>105</sup> showed that daily VED with or without weekly intracavernosal papaverine (20 mg) injection for 6 months showed significant improvement in spontaneous erectile ability (VED only: 54% (14/26 patients); VED + papaverine: 65% (17/26 patients)). In addition, VED therapy has been shown to have penile length preservation effect in both non-NSRP and NSRP.<sup>92</sup> VED has also been successfully used in patients after penile prosthesis removal. Moul and Mcleod<sup>106</sup> reported 91% (10/11 patients) satisfactory erection and successful intercourse with the same or better length and circumference, compared with the previous natural erection. The authors attributed the prevention of penile scarring and shortening to the gradual resolution of corporeal scarring, with progressively improved blood flow into corporeal sinuses by the VED therapy. Lue and El-Sakka<sup>107</sup> have reported that chronic intermittent stretching with VED successfully lengthened the shortened penis due to severe Peyronie's disease after venous grafting in three patients. Lastly, anecdotal evidence from Oakley *et al.*<sup>11</sup> indicated that VED alone improved the size of both the flaccid and the erect state of the micropallus. However, a program of penile stretching with VED showed no statistically significant difference after 6 months of therapy; although it showed a 30% rate of patient satisfaction, probably due to psychological effect.<sup>108</sup>

Some VED derivatives have been invented. Gus'kov<sup>109</sup> in 2003 reported a combination of the vacuum effect along with vibration—the vibrovacuum fallostimulator named 'Sanos'. The author claimed that this new device reduced the exposure of the penis to the vacuum effect, thus preventing edema, hemorrhage and necrosis of the penile skin. He also claimed that this had an efficacy rate of 92.1% among 330 patients; however, no further confirmatory report has been found. Another version of VED is the EROS-CTD (Clitoral Therapy Device; UroMetrics Inc., St Paul, MN, USA), the only FDA approved mechanical treatment for female sexual dysfunction. This device causes clitoral vascular engorgement using a vacuum system.<sup>110</sup> Billups *et al.*<sup>111</sup> reported that a 3-month Eros therapy improved sensation (90%), vaginal lubrication (80%), orgasm (55%), and sexual satisfaction (80%) without adverse effects in 20 women with female sexual dysfunction. Wilson *et al.*<sup>112</sup> confirmed the results in 10 women with female sexual dysfunction. Schroder *et al.*<sup>113</sup> found that the Eros therapy significantly improved patients' arousal, lubrication, orgasm and desire among 11 women with cervical cancer treated with radiation therapy and sexual arousal and/or orgasmic disorder. Billups *et al.* showed similar results in terms of vaginal lubrication (92%) and orgasm (62%) for 13 women with female sexual dysfunction and diabetes.<sup>114</sup> Many women in this study also noted that after

using the Eros therapy several times per week over a 2- to 3-month period, they continued to have improved vaginal lubrication, genital sensation or orgasms even without the device.

## Future prospects

VCD therapy is an established alternative for ED therapy when PDE<sub>5</sub>I therapy failed, contraindicated or became unaffordable. As a variation of VCD, VED has been expanding to more urological conditions that are difficult to manage. The most striking use of VED is penile rehabilitation after RP or other pelvic injuries. As data are increasingly being accumulated from clinical and basic research, the movement is gaining support to establish VED therapy as the centerpiece of PR protocol for post-prostatectomy/pelvic injuries. VT in general will be an indispensable modality in the urological armamentarium for the management of ED and related conditions.

## Conflict of interest

The authors declare no conflict of interest.

## References

- Montague DK, Barada JH, Belker AM, Levine LA, Nadig PW, Roehrborn CG *et al*. Clinical guidelines panel on erectile dysfunction: summary report on the treatment of organic erectile dysfunction. The American Urological Association. *J Urol* 1996; **156**: 2007–2011.
- King J. *Contemporary Treatment of ED*. Streight and Douglass: Indianapolis, 1874; 384.
- Lederer O. Specification of letter patent. US patent No. 1,225,341. May 8 1917.
- Sell FW. Erector. US patent No. 2,874,698. Feb 24 1959.
- Wilson FM. Apparatus for obtaining artificial erection. US patent No. 3,744,486. July 10 1973.
- Osbon GD. Erection aid device. US patent No. 4,378,008. March 29 1983.
- Lewis RW, Witherington R. External vacuum therapy for erectile dysfunction: use and results. *World J Urol* 1997; **15**: 78–82.
- Nadig PW, Ware JC, Blumoff R. Noninvasive device to produce and maintain erection-like state. *Urology* 1986; **27**: 126–131.
- Witherington R. Vacuum constriction device for management of erectile dysfunction. *J Urol* 1989; **141**: 320–322.
- Lue TF. Editorial comment on Clinical experience of vacuum tumescence enhancement therapy for impotence from Int J Impot Res 1990; **1** (suppl 2): 191–196. *J Urol* 1991; **145**: 1112.
- Oakley N, Allen P, Moore KTH. Vacuum devices for erectile impotence. In: Carson C, Kirby R, Goldstein I (eds). *Textbook of Erectile Dysfunction*. Isis Medical Media Ltd: Oxford, 1999. pp. 371–381.
- Hakim LS, Munarriz RM, Kulaksizoglu H, Nehra A, Udelson D, Goldstein I. Vacuum erection associated impotence and Peyronie's disease. *J Urol* 1996; **155**: 534–535.
- Salvatore FT, Sharman GM, Hellstrom WJ. Vacuum constriction devices and the clinical urologist: an informed selection. *Urology* 1991; **38**: 323–327.
- Marmar JL, DeBenedictis TJ, Prais DE. Penile plethysmography on impotent men using vacuum constrictor devices. *Urology* 1988; **32**: 198–203.
- User survey report 1995. *Data on File*. Osbon Medical Syatems: Augusta, GA, USA, 1995.
- Sidi AA, Becher EF, Zhang G, Lewis JH. Patient acceptance of and satisfaction with an external negative pressure device for impotence. *J Urol* 1990; **144**: 1154–1156.
- Broderick GA, McGahan JP, Stone AR, White RD. The hemodynamics of vacuum constriction erections: assessment by color Doppler ultrasound. *J Urol* 1992; **147**: 57–61.
- Bosshardt RJ, Farwerk R, Sikora R, Sohn M, Jakse G. Objective measurement of the effectiveness, therapeutic success and dynamic mechanisms of the vacuum device. *Br J Urol* 1995; **75**: 786–791.
- Tan HL. Economic cost of male erectile dysfunction using a decision analytic model: for a hypothetical managed-care plan of 100 000 members. *Pharmacoeconomics* 2000; **17**: 77–107.
- Montague DK, Jarow JP, Broderick GA, Dmochowski RR, Heaton JP, Lue TF *et al*. Chapter 1: the management of erectile dysfunction: an AUA update. *J Urol* 2005; **174**: 230–239.
- Hakim LS, Kim C, Krongrand A, Nehra A. Intracavernosal injection of prostaglandin E1 versus the vacuum erection device: a comparative analysis of the early effects on corporeal blood chemistry and blood flow. *J Urol* 1999; **161**(suppl 4): 270 (abstract 1045).
- Chen J, Mahjeesh NJ, Greenstein A. Sildenafil versus the vacuum erection device: patient preference. *J Urol* 2001; **166**: 1779–1781.
- Canguven O, Bailen J, Fredriksson W, Bock D, Burnett AL. Combination of vacuum erection device and PDE5 inhibitors as salvage therapy in PDE5 inhibitor nonresponders with erectile dysfunction. *J Sex Med* 2009; **6**: 2561–2567.
- Marmar JL, DeBenedictis TJ, Prais DE. The use of a vacuum constrictor device to augment a partial erection following an intracavernous injection. *J Urol* 1988; **140**: 975–979.
- Chen J, Godschalk MF, Katz PG, Mulligan T. Combining intracavernous injection and external vacuum as treatment for erectile dysfunction. *J Urol* 1995; **153**: 1476–1477.
- John H, Lehmann K, Hauri D. Intraurethral prostaglandin improves quality of vacuum erection therapy. *Eur Urol* 1996; **29**: 224–226.
- Segenreich E, Israilov SR, Shmueli J, Servadio C. Vacuum therapy combined with psychotherapy for management of severe erectile dysfunction. *Eur Urol* 1995; **28**: 47–50.
- Korenman SG, Viosca SP. Use of a vacuum tumescence device in the management of impotence in men with a history of penile implant or severe pelvic disease. *J Am Geriatr Soc* 1992; **40**: 61–64.
- Soderdahl DW, Petroski RA, Mode D, Schwartz BF, Thrasher JB. The use of an external vacuum device to augment a penile prosthesis. *Tech Urol* 1997; **3**: 100–102.
- Hoang An N, Romero C, Hairston JC. Vacuum constriction device: a new paradigm for treatment of erectile dysfunction. In: McVary K (ed). *Contemporary Treatment of Erectile Dysfunction*. New York: Springer, 2010 (in press).
- Limoge JP, Olins E, Henderson D, Donatucci CF. Minimally invasive therapies in the treatment of erectile dysfunction in anticoagulated cases: a study of satisfaction and safety. *J Urol* 1996; **155**: 1276–1279.
- Cookson MS, Nadig PW. Long term results with vacuum constriction device. *J Urol* 1993; **149**: 290–294.
- Segenreich E, Shmueli J, Israilov S, Raz D, Servadio C. Treatment of erectile dysfunction with vacuum constriction device. *Harafuah* 1993; **124**: 326–328, 392.
- Blackard CE, Borkon WD, Lima JS, Nelson J. Use of vacuum tumescence device for impotence secondary to venous leakage. *Urology* 1993; **41**: 225–230.
- Meinhardt W, Lycklama à Nijeholt AA, Kropman RF, Zwartendijk J. The negative pressure device for erectile disorders: when does it fail? *J Urol* 1993; **149**: 1285–1287.
- Vrijhof HJ, Delaere KP. Vacuum constriction devices in erectile dysfunction: acceptance and effectiveness in patients

- with impotence of organic or mixed etiology. *Br J Urol* 1994; **74**: 102–105.
- 37 Baltaci S, Aydos K, Kosar A, Anafarta K. Treating erectile dysfunction with vacuum tumescence device: a retrospective analysis of acceptance and satisfaction. *Br J Urol* 1995; **76**: 757–760.
  - 38 Kolettis PN, Lakin MM, Montague DK, Ingleright BJ, Ausmundson S. Efficacy of vacuum constriction device in patients with corporeal venous occlusive dysfunction. *Urology* 1995; **46**: 856–858.
  - 39 Dutta TC, Eid JF. Vacuum constriction devices for erectile dysfunction: a long-term, prospective study of patients with mild, moderate, and severe dysfunction. *Urology* 1999; **54**: 891–893.
  - 40 Kessler WO. Nocturnal penile tumescence. *Urol Clin North Am* 1988; **15**: 81–86.
  - 41 Arauz-Pacheco C, Basco M, Ramirez LC, Pita JM, Pruneda L, Raskin P. Treatment of diabetic impotence with a vacuum device: efficacy and effects on psychological status. *Am J Med Sci* 1992; **303**: 281–284.
  - 42 Bodansky HJ. Treatment of male erectile dysfunction using the active vacuum assist device. *Diabet Med* 1994; **11**: 410–412.
  - 43 Seckin B, Atmaca I, Ozgok Y, Gokalp A, Harmankaya C. External vacuum device therapy for spinal cord injured males with erectile dysfunction. *Int Urol Nephrol* 1996; **28**: 235–240.
  - 44 Turner LA, Althof SE, Levine SB, Tobias TR, Kursh ED, Bodner D et al. Treating erectile dysfunction with external vacuum devices: impact upon sexual, psychological and marital functioning. *J Urol* 1990; **144**: 79–82.
  - 45 Turner LA, Althof SE, Levine SB, Bodner DR, Kursh ED, Resnick MI. External vacuum devices in the treatment of erectile dysfunction: a one-year study of sexual and psychosocial impact. *J Sex Marital Ther* 1991; **17**: 81–93.
  - 46 Rivas DA, Chancellor MB. Complications associated with the use of vacuum constriction devices for erectile dysfunction in the spinal cord injured population. *J Am Paraplegia Soc* 1994; **17**: 136–139.
  - 47 Ganem JP, Lucey DT, Janosko EO, Carson CC. Unusual complications of the vacuum erection device. *Urology* 1998; **51**: 627–631.
  - 48 Meinhardt W, Kropman RF, Lycklama à Nijeholt AA, Zwartendijk J. Skin necrosis caused by use of negative pressure device for erectile impotence. *J Urol* 1990; **144**: 983.
  - 49 LeRoy SC, Pryor JL. Severe penile erosion after use of a vacuum suction device for management of erectile dysfunction in a spinal cord injured patient: case report. *Paraplegia* 1994; **32**: 120.
  - 50 Kim JH, Carson 3rd CC. Development of Peyronie's disease with the use of a vacuum constriction device. *J Urol* 1993; **149**: 1314–1315.
  - 51 Theiss M, Hofmockel G, Frohmuller GW. Fournier's gangrene in a patient with erectile dysfunction following use of a mechanical erection aid device. *J Urol* 1995; **153**: 1921–1922.
  - 52 Hakim LS, Munarriz RM, Kulaksizoglu H, Nehra A, Udelson D, Goldstein I. Vacuum erection associated impotence and Peyronie's disease. *J Urol* 1996; **155**: 134–135.
  - 53 Jemal A, Siegel R, Ward E, Hao Y, Xu J, Murray T et al. Cancer statistics, 2008. *CA Cancer J Clin* 2008; **58**: 71–96.
  - 54 Han M, Partin AW, Pound CR, Epstein JI, Walsh PC. Long-term biochemical disease-free and cancer-specific survival following anatomic radical retropubic prostatectomy. The 15-years Johns Hopkins experience. *Urol Clin North Am* 2001; **28**: 555–565.
  - 55 Wang R. Penile rehabilitation after radical prostatectomy: where do we stand and where are we going? *J Sex Med* 2007; **4**: 1085–1097.
  - 56 Litwin MS, Hays RD, Fink A, Ganz PA, Leake B, Leach GE et al. Quality-of-life outcomes in men treated for localized prostate cancer. *JAMA* 1995; **273**: 129–135.
  - 57 Levine LA. Erectile dysfunction following treatment of prostate cancer: new insights and therapeutic options. *J Mens Health Gen* 2004; **1**: 328–333.
  - 58 Walsh PC, Marschke P, Ricker D, Burnett AL. Patient reported urinary continence and sexual function after anatomic radical prostatectomy. *Urology* 2000; **55**: 58–61.
  - 59 Stanford JL, Feng Z, Hamilton AS, Gilliland FD, Stephenson RA, Eley JW et al. Urinary and sexual function after radical prostatectomy for clinical localized prostate cancer: the prostate cancer outcome study. *JAMA* 2000; **283**: 354–360.
  - 60 Montorsi F, Bringanti A, Salonia A, Rigatti P, Burnett AL. Current and future strategies for preventing and managing erectile dysfunction following radical prostatectomy. *Eur Urol* 2004; **45**: 123–133.
  - 61 Karakiewicz PI, Bhojani N, Neugut A, Shariat SF, Jeldres C, Graefen M et al. The effect of comorbidity and socioeconomic status on sexual and urinary function and on general health-related quality of life in men treated with radical prostatectomy for localized prostate cancer. *J Sex Med* 2008; **5**: 919–927.
  - 62 Burnett AL, Aus G, Canby-Hagino ED, Cookson MS, D'Amico AV, Dmochowski RR et al. Erectile function outcome reporting after clinically localized prostate cancer treatment. *J Urol* 2007; **178**: 597–601.
  - 63 Chartier-Kastler E, Amar E, Chevallerier D, Montaigne O, Coulange C, Joubert JM et al. Does management of erectile dysfunction after radical prostatectomy meet patients' expectations? Results of a national survey (REPAIR) by the French Urological Association. *J Sex Med* 2008; **5**: 693–704.
  - 64 Giuliano F, Amar E, Chevallerier D, Montaigne O, Joubert JM, Chartier-Kastler E. How urologists manage erectile dysfunction after radical prostatectomy: a national survey (REPAIR) by the French urological association. *J Sex Med* 2008; **5**: 448–457.
  - 65 Mulhall JP, Morgentaler A. Penile rehabilitation should become the norm for radical prostatectomy patients. *J Sex Med* 2007; **4**: 538–543.
  - 66 Burnett AL. Rationale for cavernous nerve restorative therapy to preserve erectile function after radical prostatectomy: results from CaPSURE. *J Urol* 2004; **171**: 703–708.
  - 67 Fraiman MC, Eepor H, McCullough AR. Changes in penile morphometrics in men with erectile dysfunction after nerve-sparing radical retropubic prostatectomy. *Mol Urol* 1999; **3**: 109–115.
  - 68 Mulhall JP, Graydon RJ. The hemodynamics of erectile dysfunction following nerve-sparing radical retropubic prostatectomy. *Int J Impot Res* 1996; **8**: 91–94.
  - 69 Mulhall JP, Slovick R, Hotaling J, Aviv N, Valenzuela R, Waters WB et al. Erectile dysfunction after radical prostatectomy: hemodynamic profiles and their correlation with the recovery of erectile function. *J Urol* 2002; **167**: 1371–1375.
  - 70 Lim KB, DeYoung L, Brock G. Chronic PDE5 inhibitor altered cavernosal protein expression-use in identifying protein biomarker for erectile recovery post trauma? *J Sex Med* 2006; **3**(5 suppl): 383–384 (abstract OR-006).
  - 71 User HM, Hairston JH, Zelner DJ, McKenna KE, McVary KT. Penile weight and cell subtype specific changes in post-radical prostatectomy model of erectile dysfunction. *J Urol* 2003; **169**: 1175–1179.
  - 72 McVary KT, Podlasek CA, Wood D, McKenna KE. Apoptotic pathways are employed in neuropathic and diabetic models of erectile dysfunction. *J Urol* 2006; **175**(suppl): 387 (abstract 1203).
  - 73 Moreland RB. Is there a role of hypoxemia in penile fibrosis: a viewpoint presented to the society for the study of impotence. *Int J Impot Res* 1998; **10**: 113–120.
  - 74 Leungwattanakij S, Bivalacqua TJ, Usta MF, Yang DY, Hyun JS, Champion HC et al. Cavernous neurotomy causes hypoxia and fibrosis in rat corpus cavernosum. *J Androl* 2003; **24**: 239–245.
  - 75 Granchi S, Vannelli GB, Vignozzi L, Crescioli C, Ferruzzi P, Mancina R et al. Expression and regulation of endothelin-1



- and its receptors in human penile smooth muscle cells. *Mol Hum Reprod* 2002; **8**: 1053–1064.
- 76 Moreland RB, Traish A, McMillin MA, Smith B, Goldstein I, Saenz de Tejada I. PGE1 suppresses the induction of collagen synthesis by transforming growth factor-beta 1 in human corpus cavernosum smooth muscle. *J Urol* 1995; **153**: 826–834.
- 77 Moreland RB, Gupta S, Goldstein I, Traish A. Cyclic AMP modulates TGF-beta-1 induced fibrillar collagen synthesis in cultured human corpus cavernosum smooth muscle cells. *Int J Impot Res* 1998; **10**: 159–163.
- 78 Saenz de Tejada I, Morouluan P, Tessier J, Kim JJ, Goldstein I, Frohrib D. Trabecular smooth muscle modulates the capacitor function of the penis. Studies on a rabbit model. *Am J Physiol* 1991; **260**: H1590–H1595.
- 79 Gontero P, Kirby R. Proerectile pharmacological prophylaxis following nerve-sparing radical prostatectomy. *Prostate Cancer Prostatic Dis* 2004; **7**: 233–236.
- 80 Lehrfeld T, Lee DI. The role of vacuum erection devices in penile rehabilitation after radical prostatectomy. *Int J Impot Res* 2009; **21**: 158–164.
- 81 Blair DA, Glover WE, Greenfield AD, Roddie IC. The increase in tone in forearm resistance blood vessels exposed to increased transmural pressure. *J Physiol* 1959; **149**: 614–625.
- 82 Greenfield ADM, Patterson GC. Reactions of the blood vessels of the human forearm to increase in transmural pressure. *J Physiol* 1954; **125**: 508–524.
- 83 Diederichs W, Kaula NF, Lue TF, Tanagho EA. The effect of subatmospheric pressure on the simian penis. *J Urol* 1989; **142**: 1087–1089.
- 84 Donatucci CF, Lue TF. The effect of chronic external vacuum device usage on cavernous artery function. *Int J Impot Res* 1992; **4**: 149–155.
- 85 Müller A, Tal R, Donohue JF, Akin-Olugbade Y, Kobylarz K, Paduch D *et al*. The effect of hyperbaric oxygen therapy on erectile function recovery in a rat cavernous nerve injury model. *J Sex Med* 2008; **5**: 562–570.
- 86 Yuan JH, Westney OL, Wang R. Design and application of a new rat-specific vacuum erectile device for penile rehabilitation research. *J Sex Med* 2009; **6**: 3247–3253.
- 87 Yuan JH, Lin HC, Berardinelli F, Li P, Paredes AM, Zhang RZ *et al*. The molecular mechanism of vacuum therapy in penile rehabilitation: a novel animal study. *J Sex Med* 2010; **7**(suppl 1): S1 (abstract 27).
- 88 Hinh P, Wang R. Overview of contemporary penile rehabilitation therapies. *Adv Urol* 2008; **2008**: 481218.
- 89 Schwartz EJ, Wong P, Graydon RJ. Sildenafil preserves intracorporeal smooth muscle after radical retropubic prostatectomy. *J Urol* 2004; **171**: 771–774.
- 90 Bannowsky A, Schulze H, van der Horst C, Hautmann S, Jünemann KP. Recovery of erectile function after nerve-sparing radical prostatectomy: improvement with nightly low-dose sildenafil. *BJU Int* 2008; **101**: 1279–1283.
- 91 McCullough AR, Levine LA, Padma-Nathan H. Return of nocturnal erections and erectile function after bilateral nerve-sparing radical prostatectomy in men treated nightly with sildenafil citrate: subanalysis of a longitudinal randomized double-blind placebo-controlled trial. *J Sex Med* 2008; **5**: 476–484.
- 92 Raina R, Agarwal A, Ausmundson S, Lakin M, Nandipati KC, Montague DK *et al*. Early use of vacuum constriction device following radical prostatectomy facilitates early sexual activity and potentially earlier return of erectile function. *Int J Impot Res* 2006; **18**: 77–81.
- 93 Köhler TS, Pedro R, Hendlin K, Utz W, Ugarte R, Reddy P *et al*. A pilot study on the early use of vacuum erection device after radical retropubic prostatectomy. *BJU Int* 2007; **100**: 858–862.
- 94 Montorsi F, Guazzoni G, Strambi LF, Da Pozzo LF, Nava L, Barbieri L *et al*. Recovery of spontaneous erectile function after nerve-sparing radical retropubic prostatectomy with and without early intracavernous injections of alprostadil: results of a prospective, randomized trial. *J Urol* 1997; **158**: 1408–1410.
- 95 Mulhall J, Land S, Parker M, Waters WB, Flanigan RC. The use of an erectogenic pharmacotherapy regimen following radical prostatectomy improves recovery of spontaneous erectile function. *J Sex Med* 2005; **2**: 532–540.
- 96 Nandipati K, Raina R, Agarwal A, Zippe CD. Early combination therapy: intracavernosal injections and sildenafil following radical prostatectomy increases sexual activity and the return of natural erections. *Int J Impot Res* 2006; **18**: 446–451.
- 97 Zippe C, Nandipati K, Agarwal A, Raina R. Sexual dysfunction after pelvic surgery. *Int J Impot Res* 2006; **18**: 1–18.
- 98 Ellis R, Smith A, Wilson S, Warmington S, Ismail T. The prevalence of erectile dysfunction in post-treatment colorectal cancer patients and their interests in seeking treatment: a cross-sectional survey in the West-Midlands. *J Sex Med* 17 August 2009; e-pub ahead of print.
- 99 Munding MD, Wessells HB, Dalkin BL. Pilot study of changes in stretched penile length 3 months after radical retropubic prostatectomy. *Urology* 2001; **58**: 567–569.
- 100 Savoie M, Kim SS, Soloway MS. A prospective study measuring penile length in men treated with radical prostatectomy for prostate cancer. *J Urol* 2003; **169**: 1462–1464.
- 101 Gontero P, Galzerano M, Bartoletti R, Magnani C, Tizzani A, Frea B *et al*. New insights into the pathogenesis of penile shortening after radical prostatectomy and the role of post-operative sexual function. *J Urol* 2007; **178**: 602–607.
- 102 Dalkin BL, Christopher BA. Preservation of penile length after radical prostatectomy: early intervention with a vacuum erection device. *Int J Impot Res* 2007; **19**: 501–504.
- 103 Bellorofonte C, Dell’Acqua S, Mastromarino G, Tombolini P, Ruoppolo M, Zaatar C. External devices: for which patients? *Arch Ital Urol Androl* 1995; **67**: 293–298.
- 104 Cecchi M, Sepich CA, Felipetto R, Viganò L, Pagni G, Minervini R *et al*. Vacuum constriction device and topical minoxidil for management of impotence. *Arch Esp Urol* 1995; **48**: 1058–1059.
- 105 Colombo F, Cogni M, Deiana G, Mastromarino G, Vecchio D, Patelli E *et al*. Vacuum therapy. *Arch Ital Urol Nefrol Androl* 1992; **64**: 267–269.
- 106 Moul JW, McLeod DG. Negative pressure devices in the explanted penile prosthesis population. *J Urol* 1989; **142**: 729–731.
- 107 Lue TF, El-Sakka AI. Lengthening shortened penis caused by Peyronie’s disease using circular venous grafting and daily stretching with a vacuum erection device. *J Urol* 1999; **161**: 1141–1144.
- 108 Aghamir MK, Hosseini R, Alizadeh F. A vacuum device for penile elongation: fact or fiction? *BJU Int* 2006; **97**: 777–778.
- 109 Gus’kov AR. Diagnosis and treatment of vascular erectile dysfunction with the vibration–vacuum penis stimulator ‘Sanos’. *Urologiia* 2003; **5**: 35–40.
- 110 Billups KL. The role of mechanical devices in treating female sexual dysfunction. *World J Urol* 2002; **20**: 137–141.
- 111 Billups KL, Berman L, Berman J, Metz ME, Glennon ME, Goldstein I. A new non-pharmacological vacuum therapy for female sexual dysfunction. *J Sex Marital Ther* 2001; **27**: 435–441.
- 112 Wilson SK, Delk 2nd JR, Billups KL. Treating symptoms of female sexual arousal disorder with the Eros-Clitoral Therapy Device. *J Gend Specif Med* 2001; **4**: 54–58.
- 113 Schroder M, Mell LK, Hurteau JA, Collins YC, Rotmensch J, Waggoner SE *et al*. Clitoral therapy device for treatment of sexual dysfunction in irradiated cervical cancer patients. *Int J Radiat Oncol Biol Phys* 2005; **61**: 1078–1086.
- 114 Billups K, Goldstein I. Vacuum-induced clitoral engorgement for treatment of female sexual dysfunction in diabetic women. *J Urol* 2002; **167**: 281 (abstract 1105).