ED1000

Erectile Dysfunction Shock Waves Therapy (EDSWT)

Updated: Mar. 2012
“Man survives earthquakes, experiences the horrors of illness, and all of the tortures of the soul. But the most tormenting tragedy of all time is, and will be, the tragedy of the bedroom.”

_Tolstoy_
Agenda

- EDSWT: From Physics to Physiology
- EDSWT: Mechanism of action – in-vitro/in-vivo models
- ED1000 system
- Treatment procedure
- Treatment strategy
- Current and future indications
Shock wave technology

From Physics to Physiology
Shock Wave Therapy Applications

<table>
<thead>
<tr>
<th>Clinical Field</th>
<th>Shock Wave Pressure Level (Bar)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urology</td>
<td>~900 Bar</td>
</tr>
<tr>
<td>Orthopedics</td>
<td>~400 Bar</td>
</tr>
<tr>
<td>Cardiology</td>
<td>~100 Bar</td>
</tr>
</tbody>
</table>

Energy Density Level (mJ/mm²)

- Stone Fragmentation: ~100 Bar
- Anti-inflammatory: ~100 Bar
- Angiogenesis: ~100 Bar

Clinical Field:
- 80's
- 90's
- 2000
- 2010

Vascular (ED)
Shock waves in medical fields

**Urology**
Lithotripsy of urinary calculosis

**Microbiology**
Reduction of bacterial growth

**Gastroenterology**
Disintegration of calcium deposits in the pancreas

**Dentistry**
Regeneration of bone loss in periodontal disease
Antibacterial effect associated with oral infections
Disintegration of calcium deposits in obstructive sialadenitis

**Traumatology and orthopedics**
Treatment of osteochondral lesions
Treatment of femoral head necrosis
Treatment of chronic plantar fasciitis
Decalcification of calcific tendonitis
Treatment of Morton's neuroma

**Wound medicine**
Treatment of chronic soft tissue wounds

**Andrology**
Treatment of organic erectile dysfunction

**Oncology**
Acceleration of drug delivery to targets in the human body
Shockwave sources

- Lightning & thunder
- Supersonic airplane
- Gun shot
Shock waves characteristics

- Positive peak pressure
- Steep rise
- Short time duration
- Low tensile wave component
- $V=1500\text{m/sec}$ in water
Shock wave effect

Shock wave

Mechanical response

Biochemical response

Clinical response

Increase in regional perfusion
Shear-stress effect on cell membrane

1. Positive pressure - Compression forces
2. Negative / Tensile pressure - Expansion forces
3. Shear stress - biomechanical force generated on the surface of the endothelium.

- Positive pressure wave
- Tensile wave
Shear stress effect

- ↑ eNOS activation
- ↑ NO production
- Relaxation of smooth muscles

Galley & Webster 2004
1. Shock wave creates mechanical pressure and tension force on the tissue.

2. Behind the pressure front, there’s a cavitation effect: formation and collapse of bubbles that produces micro jet of the secondary energy.

3. Physical forces generated by cavitation are highly localized.
Formation of cavitations

- Maximally expanded bubble
- Internal collapse
- Jet of kinetic force
Angiogenesis: Background

- Angiogenesis – formation of thin-walled endothelium-lined structures with muscular smooth muscle wall.
- **Angiogenic regulatory factors**: vascular endothelial growth factor (VEGF), fibroblast growth factors (FGF), angiopoietins (Ang), platelet-derived growth factor, angiogenin, angiotropin, hepatocyte growth factor, platelet endothelial cell adhesion molecule, and others....
- **Angiostatic factors**: angiostatin, endostatin, thrombospondin, CXC chemokines, and pigment epithelium–derived factor.
- Homeostatic conditions: a balance between these pro- and anti-angiogenic factors.
- Injury: the balance shifts toward pro-angiogenic factors to drive repair.
Main angiogenesis markers

- **Endothelial nitric oxide synthase (eNOS)** – an enzyme that catalyze production of nitric oxide (NO) in blood vessels and involved in regulating vascular function.

- **Vascular endothelial growth factor (VEGF)** - family of a growth factors involved in both vasculogenesis (formation of the embryonic circulatory system) and angiogenesis (the growth of blood vessels from pre-existing vasculature).

- **Endothelial Progenitor Cells** - population of the cells with ability to differentiate into endothelial cells, the cells that make up lining of blood vessels.
Biochemical response

VEGF and flt-1 upregulation - SW upregulates VEGF and its receptor, Flt-1, in endothelial cells in vitro and VEGF in the ischemic myocardium in vivo.

Endothelial Progenitor Cells recruitment - SW recruits systemically infused Endothelial Progenitor Cells in a rat model of chronic limb ischemia.

NO synthesis - SW causes nonenzymatic nitric oxide synthesis from L-arginine and hydrogen peroxide.

Anti-inflammatory effect - Modulation of neuronal NOS catalytic activity, NO production, NFkappaB activation, isoform NOS and TNF-alpha mRNA expression.

Vasodilation – SW reduces arterial perfusion pressure in artificially perfused rabbit kidneys with immediate increase in blood flow around the treated area.

Neovascularization – SW induces neovascularization at tendon via upregulation of endothelial nitric oxide synthesis, VEGF, and proliferating cell antigen.

Local perfusion - Myocardial perfusion in the ischemic myocardium was improved only where the SW’s were applied.
Shockwaves pre-clinical studies

In-vitro/In-vivo
### Physiological Effects

<table>
<thead>
<tr>
<th>Physiological Effect</th>
<th>Based on</th>
<th>Study Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shear stress</td>
<td>Shockwave exert a “cavitation effect” (inside and outside of cells) inducing localized stress on cell membranes that resembles shear stress</td>
<td>Maisonhaute E, Prado C, White PC. Surface acoustic cavitation understood via nanosecond electrochemistry. <em>Ultrason Sonochem</em> 2002;</td>
</tr>
<tr>
<td>VEGF and flt-1 upregulation</td>
<td>SW upregulates VEGF and its receptor, Flt-1, in endothelial cells in vitro and VEGF in the ischemic myocardium in vivo.</td>
<td>Nishida T, Shimokawa H, Oi K, Tatewaki H. Extracorporeal cardiac shock wave therapy markedly ameliorates ischemia-induced myocardial dysfunction in pigs in vivo. <em>Circulation</em> 2004</td>
</tr>
<tr>
<td>Local perfusion increased</td>
<td>Myocardial perfusion in the ischemic myocardium was improved only where the SW's were applied.</td>
<td>Fukumoto Y, Shimokawa H et al 2006</td>
</tr>
</tbody>
</table>
Stimulation of NO synthase activity

• Extracorporeal shockwaves at a low energy density increase nitric oxide synthase (NOs) activity and nitric oxide (NO) production in the rat glioma cells.

• NOs is a shockwave “dose”- dependent

• Maximum level of eNOS activation at an energy level of 0.03 to 0.11

Ciampaa AR et al. Nitric oxide mediates anti-inflammatory action of extracorporeal shock waves. 2005
Recruitment of circulating EPCs to ischemic and non-ischemic tissue

- Adductor muscles of hind limb of nude rat: ischemic limb (treated with SW’s), non-ischemic limb (treated with SW), control limb (no ischemia, no SW)

- Preconditioning with low-energy SW followed by injection of EPC
Recruitment of EPC (2)

Effect of SW treatment on the number of VEGF cells (chemoattractant factor for EPC) in ischemic and non-ischemic tissue

Shock wave induces neovascularization

- Rabbits: right limb (study side) treated with SW and the left limb (control side) no SW.
- Neovascularization confirmed by biopsy and angiogenic markers (VEGF, eNOS).
- Angiogenic markers following SW’s returns to baseline values after 12 wks.

Shockwaves induces neovascularization (2)

- Dogs: right limb (study side) treated with low-energy SW; left limb (control side) did not receive SW.
- Neovascularization confirmed by microscopic examination.
- Specimens: new capillary and muscularized vessels.
- Myofibroblasts: study specimens - predominantly at 8 weeks; control specimens: myofibroblasts were not seen.

<table>
<thead>
<tr>
<th>Specimens</th>
<th>Number of capillaries</th>
<th>Average (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0, 0, 0, 0, 0, 0, 0</td>
<td>0</td>
</tr>
<tr>
<td>Study (4 wks)</td>
<td>17.7, 6.7, 7.7, 37.3, 24.3, 10.3, 20.3, 14.3</td>
<td>17.33 (6.7–37.3)</td>
</tr>
<tr>
<td>Study (8 wks)</td>
<td>33, 19.3, 10, 31, 6.7, 10.3, 5, 13.7</td>
<td>16.13 (5–33)</td>
</tr>
</tbody>
</table>

*200× high power field.

<table>
<thead>
<tr>
<th>Specimens</th>
<th>Number of vessels</th>
<th>Average (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0, 0, 0, 0, 0, 0, 0</td>
<td>0</td>
</tr>
<tr>
<td>Study (4 wks)</td>
<td>2, 6, 1, 2, 4, 2, 1, 4.</td>
<td>2.75 (1–6)</td>
</tr>
<tr>
<td>Study (8 wks)</td>
<td>4, 5, 0, 8, 3, 4, 0, 5.</td>
<td>4.63 (0–8)</td>
</tr>
</tbody>
</table>

*200× high power field.
Shockwave effect on ischemia

- Rabbits: surgically excised unilateral femoral artery treated with shock wave therapy (Tx group) and not treated (control group)
- Low energy: 0.09 mJ/ mm²,
Shock wave effect on ischemia (2)

Collateral development post shock wave treatment (iliac angiography)

Capillary density post shock waves (day 28)


* Capillary / Muscle Fiber ratio
Shock wave effect on ischemia-induced myocardial dysfunction

- Pigs: Ameroid constrictor put around LCx to gradually (4 weeks) - artery occlusion without causing myocardial infarction.
- In 4 weeks SW group received shock wave therapy (SW group) and control group did not.

Nishida. Extracorporeal cardiac shock wave therapy markedly ameliorates ischemia-induced myocardial dysfunction in pigs in vivo. Circulation 2004
Shock wave effect on ischemia-induced myocardial dysfunction (2)

Enhancement of Coronary Collaterals

Control group

SW group

4 weeks post AC implantation

4 weeks post SW treatment

Nishida T Extracorporeal cardiac shock wave therapy markedly ameliorates ischemia-induced myocardial dysfunction in pigs  Circulation 2004
Shock wave effect on ischemia-induced myocardial dysfunction (3)

Left ventricle wall motion (ventriculography) : improvement of myocardial function

Control group

SW group

4 weeks post AC implantation

4 weeks post SW treatment

Nishida T Extracorporeal cardiac shock wave therapy markedly ameliorates ischemia-induced myocardial dysfunction in pigs  Circulation 2004
Shock wave effect on ischemia-induced myocardial dysfunction (4)

Improvement in LV Ejection Fraction

Sw (n=8) vs Control (n=8)

P< 0.01

Nishida. Extracorporeal cardiac shock wave therapy markedly ameliorates ischemia-induced myocardial dysfunction in pigs in vivo. Circulation 2004
Shock wave effect on ischemia-induced myocardial dysfunction (5)

Improvement of Regional Myocardial Blood Flow (colored microspheres)

Nishida. Extracorporeal cardiac shock wave therapy markedly ameliorates ischemia-induced myocardial dysfunction in pigs in vivo. *Circulation* 2004
Shock wave therapy improved LVEDV, LVEDP and LVEF

Ito Y et al. Cardiac shock wave therapy ameliorates left ventricular remodeling after myocardial ischemia-reperfusion injury in pigs in vivo. Coron Artery Dis. 2010
Shock Wave Reverses Ischemia-Related LV Dysfunction and Remodeling

Study design

At day 0
- Echocardiography study for mini-pigs (n = 12)
- LAD constriction using constrictor (n = 12)

Group 1: Ischemia only (n = 6)
- At day 90: Echocardiography study
  - Without treatment
- At day 180: Echocardiography study
  - Cardiac Catheterization
  - Sacrificed and collected heart tissue

Group 2: Ischemia + ECSW (n = 6)
- At day 90: Echocardiography study
  - ECSW therapy (800 impulses, 0.09 mJ/mm)
- At day 180: Echocardiography study
  - Cardiac Catheterization
  - Sacrificed and collected heart tissue

Number of small vessels

A Normal
B Ischemia
C Ischemia + SW

Number of apoptotic nuclei

A Normal
B Ischemia
C Ischemia + SW

Fibrotic area

E Normal
F Ischemia
G Ischemia + SW

Shock Wave Reverses Ischemia-Related LV Dysfunction and Remodeling (2)
Ischemic heart + Shockwave

- Enhanced Angiogenesis
- Promoted EPC Homing
- Suppressed Apoptosis
- Preserved Mitochondrial Function
- Reduced Oxidative Stress
- Alleviated Inflammation

Increased endothelial cells in ischemic tissue
- Attenuated cardiomyocyte death
- Attenuated myocardial fibrosis
- Attenuated inflammation response

Limited Remodeling & Preserved Cardiac Function
- Mean fibrotic area ↓
- Cx43 protein expression ↑
- LVESV ↓
- LVESD ↓
- LVEDP ↓
- LVEF ↑
- FS ↑
Summary

• SW’s induces local angiogenesis
• The effect is dose dependant
• The effect is energy dependant
• The effect is local and focused
• Angiogenesis factors released into the blood following SW’s have a limited life span
Summary

• SW’s improve myocardial ischemia
• SW’s reduce the remodeling effect following myocardial ischemia
• SW’s reduce the impact of ischemia-reperfusion injury
• SW’s reverses ischemia-related left ventricle dysfunction
Low-energy shockwave effect

Shock wave

- Mechanical response
  - Shear stress
  - Cavitation

- Biochemical response
  - Endothelial Nitric Oxide Synthase (eNOs)
  - Vascular Endothelial Growth Factor (VEGF)
  - Endothelial Progenitor Cells (EPC)

- Clinical response
  - Short-term response
    - Vasodilatation
  - Long-term response
    - Angiogenesis
    - Neovascularization

Increase in local perfusion
BREAK
Shock wave technologies

Electrohydraulic / Ellipsoid
Spark Gap technology: uses a spark plug to generate a shockwaves focused by an ellipsoid reflector.

Electromagnetic / Flat Coil (Lens)
Uses a cylindrical coil arrangement of an electromagnetic generator and a parabolic reflector to focus the shockwaves.

Piezoelectric / Sphere
Ceramic elements are lined on a reflector dish with shock waves generated by an electric pulse. Shock waves focused by thousands of small crystals in the applicator head.
Ellipsoid Advantages

Each shock wave travels the same distance from F1 to F2, and reaches F2 at the same time.

Semi ellipsoid of the Cardiospec Shock Wave Applicator
Shock waves - principles

Generated by electro-hydraulic effect:
- High voltage creates electric spark discharge
- The water vaporizes and creates an explosion
- Generating high energy Shock Waves
- Reflected by the Semi-Ellipsoid
- Focused onto the treatment area
Shock wave therapeutic coverage

Shock wave frequency (Fourier Transform)

Pulse Wave Length: 4 μs

Pick Frequency: 300KHz (100KHz - 1 MHz)
Shock waves vs. ultrasound waves

- Mechanical Pressure (shear stress)
- High Energy (100 Mpa)
- Not reflected by ribs (larger acoustic window)
- Focused waves

- Heating effect (protein denaturation)
- Low Energy (<0.5 Mpa)
- Reflected by ribs (limited acoustic window)
- Diffused wave
Attenuation of SW’s in tissue

<table>
<thead>
<tr>
<th>Material</th>
<th>Density (kg/m³)</th>
<th>Velocity of sound (m/s)</th>
<th>Acoustic Impedance (kg / m²·s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>1.2</td>
<td>330</td>
<td>0.0004 x 10⁶</td>
</tr>
<tr>
<td>Water</td>
<td>1000</td>
<td>1437</td>
<td>1.44 x 10⁶</td>
</tr>
<tr>
<td>Fat</td>
<td>970</td>
<td>1480</td>
<td>1.44 x 10⁶</td>
</tr>
<tr>
<td>Muscle</td>
<td>1060</td>
<td>1570</td>
<td>1.66 x 10⁶</td>
</tr>
<tr>
<td>Bone:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortical</td>
<td>1700</td>
<td>3600</td>
<td>6.12 x 10⁶</td>
</tr>
<tr>
<td>Cancellous</td>
<td>~1000</td>
<td>1450</td>
<td>1.45 x 10⁶</td>
</tr>
</tbody>
</table>

- Body tissues has low attenuation effect
Extracorporeal Shockwave Myocardial Revascularization

Cardiospec system
ESMR principle

• Low-energy, focused shock waves
• Energy density: 0.09 mJ/mm²
• Echo-guided treatment
• ECG-gated (R-wave)
System components

- Control Panel
- Ultrasound probe holder
- Shock Wave Applicator
- Treatment table
- Power Cabinet
- ECG monitor
System components

- Shock wave applicator with inflated membrane
- Echo transducer
- Positioning device (X- and Y-axis movement)
ESMR treatment set-up
Case Study

Evaluation by SPECT
Extracorporeal Shockwave Myocardial Revascularization

Current clinical indications
ESMR clinical aim

- Induce local angiogenesis at myocardial ischemic areas using low intensity, non invasive, focused shock waves
- Treatment option for patients who no longer benefit from current revascularization methods
Treatment of end-stage Coronary Artery Disease


Pre - Treatment

Pre: CCS class IV

Post: CCS class III

*Courtesy of Prof. R. Erbel, Essen, Germany
Treatment of 3-Vessel Disease

Patient: 75 yo male, DM, HPT, Dyslipidaemia, Smoking, 3-vessel disease, prior PCI, CABG

Pre - Treatment

Post – Treatment 6-month follow-up

CCS class: 3-2, SAQ: 41.17 – 52.06
EST duration: 6.18-9.37, METS: 5.7-5.3
LVEF: 24-46, TDI: 45-18

*Courtesy of Prof. Wan Azman, Kuala Lumpur, Malaysia
Improvement of regional myocardial blood flow (assessed by PET scan)

P=0.04

Faber et al. 2010
Chronic Heart Failure Study

Minnesota Living with Heart failure

Nitroglycerin doses per week

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin doses per week</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Left ventricular ejection fraction

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>3 months</th>
<th>6 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF, %</td>
<td>35.4</td>
<td>27.8</td>
<td>28.2</td>
</tr>
</tbody>
</table>

Vasyuk et al. 2010
Long-term results

NTG uptake/Hospitalization - 1 yr follow-up

Results 5 years follow up

CCS 5 years FU

p=0.002

p < 0.001

n.s.

n=23

n=21

Alunni, 2011

Gutersohn A, 2006
## Increase of Exercise Tolerance Capacity

<table>
<thead>
<tr>
<th>Study</th>
<th>Improvement (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yaakob Z, 2011</td>
<td>25%</td>
</tr>
<tr>
<td>Panaeva S, 2010</td>
<td>28%</td>
</tr>
<tr>
<td>Fu M, 2010</td>
<td>29%</td>
</tr>
<tr>
<td>Leibovitz D, 2010</td>
<td>33%</td>
</tr>
<tr>
<td>Vasyuk Y, 2010</td>
<td>30%</td>
</tr>
<tr>
<td>Vainer J, 2010</td>
<td>35%</td>
</tr>
<tr>
<td>Faber L, 2008</td>
<td>13%</td>
</tr>
<tr>
<td>Koltunov I, 2008</td>
<td>40%</td>
</tr>
<tr>
<td>Samad A, 2008</td>
<td>14%</td>
</tr>
<tr>
<td>Lyadov K, 2006</td>
<td>63%</td>
</tr>
<tr>
<td>Naber CK, 2008</td>
<td>64%</td>
</tr>
</tbody>
</table>

*Mean improvement (*) 34%

(*) Different analysis methods  (**) n=240
Decrease of Weekly Nitrate Intake

Panaeva S, 2010 86%
Nikonenko A, 2010 71%
Zuoziene G, 2010 92%
Vasyuk Y, 2010 64%
Vainer J, 2010 82%

Mean improvement 79%
Improvement of Myocardial Perfusion

Yaakob Z, 2011 39.43%
Alunni G, 2010 21%
Vasyuk Y, 2010 43%
Faber L, 2008 20%
Takayama T, 2008 19.5%
Naber CK, 2008 53%

(*): Different analysis methods  (**) n=114
Safety – Troponin I levels (n=78)

Summary

- Extracorporeal Shockwave Myocardial Revascularization
- Non-invasive treatment for patients with myocardial reversible ischemia untreatable by conventional methods
- Triggering local angiogenesis in the treatment area
- Painless, safe treatment without reported side effects
- www.esmr.info
Shock wave theory of operation

- Shockwaves are being successfully used around the world since 2005 to treat reversible ischemic tissues in the heart, thus inducing neovascularization and developing new collaterals. This improves the volume of blood flow into the tissue.

- The same modality is used for treatments for vascular-based Erectile Dysfunction problems.
Rational behind EDSWT

- Extracorporeal shockwave treatment promote endothelial cell precursors.
- Extracorporeal shockwave treatment enhance expression of vascular endothelial growth factors and its receptor.
- Extracorporeal shockwaves clinically induces neovascularization which improves tissue perfusion.
- ED is a disease of the endothelium.
- Extracorporeal shockwave treatment can be used for treatments of vascular-based Erectile Dysfunction.
ED1000 system
The ED1000 system

Low intensity Extracorporeal Shock waves especially designed for the treatment of Erectile Dysfunction
Device Description

Dimensions: height: 740 mm x width: 775 mm x depth: 410 mm; Weight: 35 kg
Device Description

- "Umbilical Cord"
- SWA supporting Arm Port
- Mains Connection, Footswitch Connection & Master Switch
Control Panel

ON/OFF Switch

LCD Touch-Screen & Display
Treatment Screen

- High-Voltage Indicator
- Shockwave Indicator
- General Warning
- System Status Indicator

Operate Key
Applicator Indicator
Time & Date Display

Return Key
Service Screen
Information Key
Number of Shocks selection

- Treatment Counter Reset
- Activation Countdown Key
- Numerical Keypad
Number of Shocks selection

- **Down Counter display**
- **Countdown Value**
- **Number of Shocks selected**
Messages - Applicator is not Properly Connected
Messages-SWA Applicator Out of Range and must be Replaced
Messages-Incorrect SWA Applicator
Messages Hardware or Operational Software Failure!
Information Screen

- Number of Shocks performed by the Device
- ED1000 Serial Number
- Number of Shocks left in the SWA
- SWA Serial Number
- SWA Expiration date
Service Screen requires password!
Press Return Key to exit.
## System features & benefits

<table>
<thead>
<tr>
<th>“Plug &amp; play”</th>
<th>No special electrical requirements.</th>
</tr>
</thead>
<tbody>
<tr>
<td>User-friendly</td>
<td>▪ Touch screen control panel.</td>
</tr>
<tr>
<td></td>
<td>▪ Shockwaves countdown.</td>
</tr>
<tr>
<td>Portable</td>
<td>▪ Compact</td>
</tr>
<tr>
<td></td>
<td>▪ Easy to move</td>
</tr>
<tr>
<td></td>
<td>▪ Easy to store.</td>
</tr>
<tr>
<td>Cost-effective</td>
<td>▪ Quick operator training.</td>
</tr>
<tr>
<td></td>
<td>▪ Low cost maintenance.</td>
</tr>
<tr>
<td></td>
<td>▪ Applicator is the only consumable</td>
</tr>
<tr>
<td>Applicator</td>
<td>When needed, applicator replaced entirely</td>
</tr>
</tbody>
</table>
Treatment method
Treatment methods

- EDSWT is applied on the penile shaft and corpus.
- Each treatment included a 3-minute application of 300 shock waves in 5 different anatomical sites.
Technique

Shaft

Crura
Penis Anatomy
Penis Anatomy
Treatment of the shaft

1. Stretch the penis.
2. Firmly attach shockwave applicator to the treatment location with the SWA center perpendicular to the shaft.
3. Apply 300 shocks at three locations across the shaft (below the gland and above the base).
4. Use generous amount of coupling ultrasound gel.
Treatment of the crura

1. Locate the pubic bone of the patient.
2. Deliver 300 shocks beneath the pubic bone with the SWA center toward the crus as indicated on the picture.
3. Repeat the procedure on the other side with additional 300 shocks.
4. Use generous amount of coupling ultrasound gel.
Treating the Cruz: a look from the side
Treating the Cruz: patient lie down
Treatment protocol

Per Patient:
- 2x6 treatments drg. 9 weeks
- 5 treatment zones
- 300 shocks per zone

18,000 shock

Total Treatment Time:
20 minutes
Air bubbles

- During shock wave discharge air bubbles are created and accumulate within the SWA membrane.

Air diminishes propagation of shockwaves in the body.

Air can be detected by the following means:

- Sound – with air accumulation the shockwaves sound becomes “metallic”
- Sight – air bubble is visible when turning the SWA upside down
<table>
<thead>
<tr>
<th>Advantage</th>
</tr>
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<tbody>
<tr>
<td>• Novel treatment option for patients with ED offering real cure</td>
</tr>
<tr>
<td>• No use of medications</td>
</tr>
<tr>
<td>• No reported side effects</td>
</tr>
<tr>
<td>• Use on demand</td>
</tr>
<tr>
<td>• Therapeutic angiogenesis using shockwaves</td>
</tr>
</tbody>
</table>
ED-1000 Installations