The old wave

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Introduction
In 1788, Charles Kite, a member of the Royal Humane Society of London, an organisation devoted to salvaging persons seemingly dead, described the use of electricity to revive a three-year-old child who was taken for dead after falling out of a window.

An “apothecary” was sent for, who could do nothing. Electrical resuscitation (see Figure 1) was attempted by a Mr Squires, who “with the consent of the parents, very humbly tried the effects of electricity. At least 20 minutes had elapsed before he could apply the shock, which he gave to various parts of the body without any apparent success. At length, on transmitting a few shocks through the thorax, he perceived a small pulsation. Soon after, the child began to breathe, though with great difficulty. About 10 minutes later, she vomited. A kind of stupor remained for some days, but the child was restored to perfect health and spirits in about a week”.

Kite commented: “Do (these examples) not plainly point out, that electricity is the most powerful stimulus we can apply? And are we not justified in assuming that if it is able so to excite the action of the external muscles so powerfully, that it will be capable of reproducing the motion of the heart, which is infinitely more irritable, and by that means accomplish our great desideratum, the renewal of the circulation?”

Twentieth century advancements in defibrillation
Further experiments on “faradisation” of the heart were conducted by two physiologists, J-L Prevost and F Batelli, from the University of Geneva, Switzerland (see Figures 2a and 2b). They discovered that, while a weak stimulus can produce fibrillation, a stimulus of higher strength applied to the heart could arrest ventricular fibrillation and restore normal sinus rhythm. This discovery was made in 1899. Unfortunately, unlike the discovery of the contemporary electrocardiogram, defibrillation did not enjoy similar attention and success.

Prevost and Batelli's discovery was confirmed and advanced by subsequent work in many countries, most prominently by the research laboratory of Carl J Wiggers (see Figure 3a).
from the Western Reserve University in Cleveland, Ohio. Using a cinematograph, a state-of-the-art experimental methodology of his time, Wiggers was able to advance the original observations of Vulpian, describing several stages of ventricular fibrillation produced by a stimulus, which now are known as Wiggers Stage I, Wiggers Stage II, etc.

Wiggers provided the first mechanical explanation of the induction of ventricular fibrillation within the framework of the concept of the vulnerable window (see Figure 3b). He also perfected the defibrillation procedure in an animal model of defibrillation.

Wiggers' work in the Department of Physiology of Western Reserve University was well known to thoracic surgeon, Claude S Beck, from the University Hospitals in Cleveland, which are adjacent to the Western Reserve University. In 1947, Dr Beck successfully applied defibrillation therapy and saved the first human life by this method. His success triggered the immediate acceptance of this method by the clinical community and started widespread basic and clinical research of fibrillation and defibrillation (see Figure 4).

The work of Prevost and Batelli was independently continued by the Russian physiologists, NA Negovsky and NL Gurvich in Moscow (see Figure 5). Gurvich was trained by LS Schtern, the director of the Institute of Physiology in Moscow, who graduated from the University of Geneva and was Prevost's associate for many years. Gurvich made many important discoveries and advancements in defibrillation, including the biphasic waveform and use of a capacitor for shock delivery. He also introduced the stimulatory theory of defibrillation.

In 1967 in Belfast, Northern Ireland, Dr Frank Pantridge was the first to demonstrate how victims of sudden cardiac death could be successfully resuscitated outside of the hospital environment. He did so by initiating a unique programme. He sent an ambulance staffed with a resuscitation team to people who were suffering chest pain. An AC defibrillator, powered by two 12-volt car batteries using a converted static inverter, was mounted in the ambulance. In this experimental vehicle, the first 10 patients suffering cardiac arrest were all successfully resuscitated. As the project continued and greater numbers were available for study, the Belfast investigators noted that the time it took from the onset of cardiac arrest to the first defibrillation attempt was the single most significant factor in determining a successful resuscitation.
The innovative Belfast programme was the catalyst for the development of advanced life support (ALS) throughout the USA, and in fact, the world. But despite this legacy, during the past three decades, emergency medical systems have struggled to optimise sudden cardiac arrest survival rates. The national average remains well below 10%, and in many large cities, below two per cent. The advent of automatic defibrillators provided a potential solution. The new technology allowed defibrillation to be performed by a wide variety of individuals with diverse backgrounds and training.

First emergency medical technician-paramedics, then fire fighters, and now police, have all have shown they can improve sudden cardiac death survival rates using well-designed systems. More recently, improved survival rates on airlines and in casinos demonstrated that other categories of non-traditional responders can be effective. All the successful programmes reconfirm Pantridge’s original observation: time to defibrillation is the single most important factor in improving save rates.

What’s next? Since the majority of cardiac arrests occur in the home, the obvious new frontier is home defibrillation. As defibrillators become smaller, less expensive and easier to use, the once futuristic idea of having home defibrillators is no longer idle fancy.

Actually, initial programmes in home defibrillation began more than 15 years ago. In 1985, Mickey Eisenberg, a renowned researcher from the University of Washington, wrote an editorial entitled Automatic external defibrillation: bringing it home. In this editorial, and a subsequent one published in 2000, he raised many insightful questions. He concluded his 1985 editorial by asking: “Will this sort of defibrillation be in the hands of everyone, or will only a few wield its power?” Small, easy-to-use automatic defibrillators will soon rival the cost of home computers. And just as it becomes increasingly rare to find a household without a computer, eventually it may be equally difficult to find one that does not consider a defibrillator to be a basic first-aid tool; no less important than a smoke detector or fire extinguisher.

We know with certainty that defibrillators save lives when properly applied soon after the onset of ventricular fibrillation. There are still important academic questions to be answered by evidence-based research: What is the cost-benefit ratio compared to other health interventions? How do we optimise training, education, and continuing education? What locations and methods of deployment bring the greatest benefits at the lowest cost?

But with or without additional research, market forces and media attention are already prompting adoption of this new, exciting technology. Individuals can buy defibrillators online through Amazon.com, and they will soon become available for easy purchase from other internet sources, and even from your local supermarket. Commercial messages espousing their value are starting to appear in targeted markets and will begin to proliferate, particularly as the emergence of less expensive units makes personal defibrillators an affordable option. It’s conceivable that many individuals will own their own defibrillator before these medical devices become routinely available at shopping malls, restaurants, churches, physicians’ offices, health clubs, golf courses and other potentially high-risk locations. While academic issues are debated, the next frontier is clear. What started from Pantridge’s ideas will soon be adopted by the Smiths, the Joneses, and perhaps you.

**An introduction to biphasic technology**

Pioneered in the 1950s, defibrillation technology has remained largely unchanged for the past several decades. Without an effective choice, clinicians and professional rescuers, without concern for the side-effect profile of high-energy defibrillation, shocked countless numbers of patients. The migration of biphasic technology from the world of implantables to external defibrillation challenges convention.

The growing body of evidence demonstrates that external biphasic defibrillation is at least as effective, at lower energy levels, as conventional high-energy monophasic technology. Furthermore, the commercial availability of biphasic external defibrillators compels examination of the potential side-effect profile of higher energy systems.

Defibrillation shocks are typically characterised by the amount of energy delivered (e.g. 200, 300 or 360 joules). Alternatively, they can be described as a discharge waveform that plots electrical current (amperes) or voltage (volts) delivered versus time of delivery.

Defibrillation waveforms are generated from the discharge of energy (stored on a capacitor) through a patient. The value of the capacitor, discharge path, and elements of that path determine the shape of the waveform. Waveform shapes are used to classify defibrillation technology.

Waveforms describe the delivery of energy, or current, as a factor of time. It is worthy to note that while we may choose an energy setting in joules, it is actually the resulting current that defibrillates or cardioverts.

Using energies ranging up to 360 J, monophasic defibrillators can deliver upwards of 60 amps (see Figure 6). A body of data is accumulating that shows substantial post-shock myocardial dysfunction results from high-energy defibrillation.

**Biphasic truncated exponential (BTE) waveform**

Figure 7 depicts a typical biphasic waveform. The energy delivery occurs in two phases. The first phase, seen as the positive waveform deflection, is indicative of current flow from the sternum to apex paddles.
The second phase, depicted by the negative deflection, indicates a reverse direction.

Offering the promise of smaller size and extended battery life, the biphasic truncated exponential (BTE) waveform (Figure 8) was originally developed for the implantable cardiac defibrillator where impedance variations are a virtual non-factor. When used for external defibrillation, a passive impedance compensation occurs. This is seen in the waveform as changes to its shape. In other words, the duration and amplitude vary.

Randomised trials that examined application of the BTE waveform to external defibrillation have found that they generally match the clinical performance of monophasic technology at lower energy levels.

Rectilinear biphasic waveform

The first attribute is an initial phase that reduces peak currents by essentially delivering a constant current. This is achieved by controlling the total impedance of a defibrillation circuit, that is, the impedance of the patient and of the defibrillator. Where patient impedance is high, a series of digitally controlled resistors lowers the defibrillator resistance to maintain the constant current flow. Conversely, when it is low, the defibrillator resistance is raised.

The second attribute is the fixed duration of each of the phases. Regardless of patient impedance, the first and second phases are always six and four milliseconds, respectively. The importance of phase duration with regard to the performance of biphasic waveforms is well established in the clinical data. Several studies have demonstrated that as phasic duration changes, so too does the performance of the waveform. Generally speaking, fixed waveforms yield more consistent performance.

Comparative peak currents

Current is a critical aspect of defibrillation. While it is average current that defibrillates, studies have found that high peak currents result in myocardial dysfunction.

Defibrillation waveform overlays

Figure 9 is an overlay of the three waveforms we have reviewed. The monophasic waveform is illustrated in red, the biphasic truncated exponential is in yellow, and the rectilinear biphasic is shown as white. While the BTE waveform has a lower peak current than the monophasic waveform, the peak current for the rectilinear biphasic waveform is 65% lower than that of the monophasic technology.

Effect of patient impedance

Figure 10 shows the differing response of the BTE and RBW waveforms to patient impedance. Whereas the shape
of BTE waveforms change in the face of varying patient impedance levels, the RBW exhibits stability in both shape and duration. Stability of the waveform is important for optimal clinical results.

Early work demonstrated variability in the waveform duration effects, the defibrillation threshold, and ultimately effectiveness. Perhaps most importantly, it showed that some biphasic waveforms perform worse than conventional monophasic technology.

This continues to demonstrate that all biphasic waveforms are not the same. It further suggests that they need to be viewed in light of the clinical data.

**Waveform responses to impedance**

A prospective, multicentre trial randomised 184 patients undergoing implantable cardioverter defibrillator implantation or study into either a monophasic or biphasic arm. The monophasic patients were defibrillated with escalating energy levels of 200-300-360 J. The biphasic patients received escalating levels of 120-150-170 J.

The first-shock efficacy of 120 J rectilinear biphasic was better than that of 200 J monophasic. The single biphasic patient who was not defibrillated with the first shock was successfully converted at 150 J. The patients who failed at 200 J monophasic were successively defibrillated with energy up to 360 J.

The investigators looked at the efficacy of the rectilinear biphasic waveform in the difficult-to-defibrillate subset of high impedance patients. Whereas the first-shock efficacy was similar for lower impedance patients (< 70 W), the rectilinear biphasic waveform was dramatically better for higher impedance patients (> 70 W).

Success with this particularly tough rhythm encouraged investigators to evaluate the rectilinear biphasic waveform on a equally daunting rhythm.

**Conclusion**

Despite the relatively recent introduction of biphasic waveforms into external defibrillators, some conclusions can be safely drawn. Firstly, the data demonstrate that biphasic waveforms have at least the same first shock efficacy as monophasic waveforms. They have the advantage of doing so at substantially lower energy levels. In fact, biphasic waveforms designed specifically for transthoracic defibrillation may promise performance that surpasses that of monophasic technology.

Secondly, and perhaps most importantly, we need to recognise that all biphasic waveforms are not the same. Their peak currents and response to patient impedance greatly influence the clinical efficacy. So, as one considers the adoption of biphasic defibrillation, the clinical data generated for each type must be considered.

**References are available on request**

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Basic Life Support for Healthcare Providers
(Agent and Child)

Hazard?
Ensure scene is safe

Hello?
Check Responsiveness

Help!
Call for assistance and Defibrillator/AED

A
Open Airway
Remove visible foreign material
Look for adequate breathing

Breathe
Give 2 effective (chest rising) breaths at 1 breath/second
(with 02 if available),
Feel for pulse for up to 10 seconds.
Is a definite pulse present?

Compressions
Compress chest at a rate of 100/min (almost 2 compressions/second)
Push hard / Push fast / Ensure full chest recoil / Minimize interruptions
CPR Ratios: 1-Rescuer = 30:2 and 2–Rescuers (Child) = 15:2
Continue until Defibrillator/AED available and ready

D
If time from collapse > 5 minutes without CPR, first do 2 minutes of CPR before analysing

Analyse Rhythm

Shockable
(VF/Pulseless VT)
Give 1 Shock
Biphasic: 120-360 J (4 J/Kg)
Monophasic - 360 J (4 J/Kg)
Immediately resume CPR for 2 minutes

Non-SHockable
(PEA/Asystole)
Immediately resume CPR for 2 minutes

If safe to do so:
Treat illnesses or injuries as necessary
(Aspirin / Inhaler / Auto-injector)
Get assistance if needed
Reassess continuously

Place in recovery position
Check for continued breathing
Reassess continuously

Continue Rescue breathing:
- Adult: 10/min
- Child: 12-20/min
Reassess continuously

If time from collapse > 5 minutes without CPR, first do 2 minutes of CPR before analysing

Shockable
(VF/Pulseless VT)
Give 1 Shock
Biphasic: 120-360 J (4 J/Kg)
Monophasic - 360 J (4 J/Kg)
Immediately resume CPR for 2 minutes

Non-SHockable
(PEA/Asystole)
Immediately resume CPR for 2 minutes

After 2 min of CPR, if organized electrical activity returns, check pulse:
- If present – provide post-resuscitation care
- If absent, continue CPR

Do not interrupt chest compressions unless absolutely necessary

Resuscitation Council of Southern Africa
www.resuscitationcouncil.co.za
# Advanced Life Support for Healthcare Providers (Adult and Child)

**Hazards?**
Ensure scene is safe

**Hello?**
Check Responsiveness

**Help!**
Call for assistance and Defibrillator/AED

**Open Airway**
Remove visible foreign material
Look for adequate breathing

**Breathe**
Give 2 effective (chest rising) breaths at 1 breath/second (with 02 if available).

**Compressions**
Compress chest at a rate of 100/min (almost 2 compressions/second)
CPR Ratio: 1-Rescuer = 30:2 and 2–Rescuers (Child) = 15:2

**Analyze Rhythm**

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**Shockable**
(VF/Pulseless VT)

- **Give 1 Shock**
  - Biphasic: 120-360J (4 J/Kg)
  - Monophasic: 360 J (4 J/Kg)

- **Immediately resume CPR for 2 minutes**

**Non-Shockable**
(PEA/Asystole)

- After 2 min of CPR, if organized electrical activity returns, check pulse:
  - If present – provide post-resuscitation care
  - If absent, continue CPR

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*Correct Contributing Causes:
- Hypoxia
- Tension Pneumothorax
- Hypovolaemia
- Tamponade
- Hyper/hypokalaemia
- Trauma
- Hyper/hyperglycaemia
- Thrombosis (Pulmonary)
- Hypothermia
- Thrombosis (Coronary)

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* Do not interrupt chest compressions unless absolutely necessary

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## Adult Emergency Arrhythmia Management Algorithm

### A. AIRWAY
Open, maintain and protect as necessary

### B. BREATHING
Administer oxygen. Ventilate if necessary

### C. CIRCULATION
Assess pulse, blood pressure & perfusion. Attach ECG monitor, pulse oximeter and vital signs monitor if available

### D. DRIP
Establish IV line with Normal Saline

### E. ECG RHYTHM
Run rhythm strip to confirm arrhythmia*. 12 lead ECG if possible.

#### Brachycardia
HR<60/min

- Assess patient

#### Narrow Complex Tachycardia
(Supraventricular Tachycardia) HR > 100/min with QRS complexes < 0.12 sec

- Assess patient

#### Wide Complex Tachycardia
(Ventricular Tachycardia) HR > 100/min with QRS complexes > 0.12 sec

- Assess patient

### Adverse Features

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain</td>
<td>SBP&lt;90mmHg</td>
</tr>
<tr>
<td>Short of breath</td>
<td>Heart failure</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>HR&lt;40/min</td>
</tr>
<tr>
<td>2° Block (Type II) or 3° Block (wide QRS)</td>
<td></td>
</tr>
</tbody>
</table>

#### Narrow Complex Tachycardia

- Atropine
  - Exclude hyperkalaemia/hypoxia/hypothermia/lead injury/heart block/heart transplant
  - 0.5mg IV every 3 min (max = 3mg)

- Vagal Maneuvres
  - Cough/Valsalva/Breathholding/Trendellumberg
  - Carotid Massage (C/I with bruits, AMI, dig tox, elderly)
  - Facial application of ice water (C/I with AMI)

- Adenosine
  - 6mg IV rapidly, then 12mg IV after 2 min, then 12mg IV again if necessary

- Amiodarone
  - 150 mg over 10 min IV (15 mg/min), then 1 mg/min infusion

- Alternatives (under cardiologist supervision):
  - β - Blockers (eg Esmolol)
  - Ca Channel Blockers (eg Verapamil) or Diltiazem

### Wide Complex Tachycardia

- Synchronised Cardioversion (Sedate if necessary)
  - Start with 100J initially (monophasic or biphasic)

- Amiodarone
  - 150 mg over 10 min IV (15 mg/min), then 1 mg/min infusion or

- Lidocaine
  - 1mg/kg IV every 5 min pm (Max=3mg/kg) and 2mg/min infusion

### Consider

- Glucagon (3mg IV, then 3mg/hr)

(The algorithm follows the assumption that the previous step was unsuccessful and the patient is deteriorating)

*NB: SPECIALIST MEDICAL ADVICE SHOULD BE SOUGHT WHENEVER POSSIBLE.

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# Paediatric Emergency Arrhythmia Management Algorithm

## A. AIRWAY
- Open, maintain and protect as necessary.

## B. BREATHING
- Administer oxygen. Ventilate if necessary.

## C. CIRCULATION
- Assess pulse, blood pressure & perfusion.
- Attach ECG monitor, pulse oximeter and vital signs monitor if available.

## D. DRIP
- Establish IV line with Normal Saline.

## E. ECG RHYTHM
- Run rhythm strip to confirm arrhythmia.
- 12 lead ECG if possible.

### Bradycardia
- HR<60/min despite effective oxygenation & ventilation.

#### Adverse Features
- Poor perfusion, Hypotension.
- Respiratory difficulty.
- Level of consciousness (Exclude hyperkalaemia/hypoxia/hypothermia/head injury/heart block).

#### Start CPR
1. Rescuer: 2 breaths: 30 compressions.
2. Rescuers: 2 breaths: 15 compressions.

#### Adrenaline (1:1000)
- 0.01 ml/kg IV/IO (0.1 ml/kg ET) every 3-5 minutes.

#### Vagal Manoeuvres
- (Except varying R-R intervals/AF)
  - Cough/Valsalva/Breathholding/Trendelenberg
  - Carotid Massage (older children)
  - Apply ice to face (infant & young child).

#### Amiodarone
- 5mg/kg IV over 20-60 min (Maximum - 300mg).

### Narrow Complex Tachycardia
- (Supraventricular Tachycardia)
- HR > 220/min (infant) > 180/min (child).

#### Adverse Features
- Poor perfusion, Hypotension.
- Respiratory difficulty.
- Level of consciousness.
- Abrupt HR change.
- P-waves absent or abnormal.

#### Vagal Manoeuvres
- (Except varying R-R intervals/AF)
  - Cough/Valsalva/Breathholding/Trendelenberg
  - Carotid Massage (older children)
  - Apply ice to face (infant & young child).

#### Amiodarone
- 5mg/kg IV over 20-60 min (Maximum - 300mg).

### Wide Complex Tachycardia
- (Ventricular Tachycardia)
- HR > 200/min with QRS complexes > 0.08 sec.

#### Adverse Features
- Poor perfusion, Hypotension.
- Respiratory difficulty.
- Level of consciousness.
- Abrupt HR change.
- P-waves absent or abnormal.

#### Synchronised Cardioversion
- (Sedate if necessary)
  - 0.5-1J/kg, then 2J/kg (monophasic or biphasic).

#### Amiodarone
- 5mg/kg IV over 20-60 min (Maximum - 300mg).

(The algorithm follows the assumption that the previous step was unsuccessful and the patient is deteriorating)

*NB: SPECIALIST MEDICAL ADVICE SHOULD BE SOUGHT WHENEVER POSSIBLE.*

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Recommended Defibrillator Energy Settings in Cardiac Arrest

(Adult and Child)

Paediatric Patients (Pre-Puberty)

*Monophasic & Biphasic Defibrillators* - 4 Joules/kg (1st and subsequent shocks)

**Adult Patients (Post-Puberty)**

*Monophasic Defibrillators* - 360 Joules (1st and subsequent shocks)

*Biphasic Defibrillators* - As per Manufacturer’s recommendations (See Table)

<table>
<thead>
<tr>
<th>Defibrillator Make</th>
<th>Distributor in SA</th>
<th>Biphasic Waveform</th>
<th>Recommended Energy Setting in Cardiac Arrest (Joules)</th>
</tr>
</thead>
<tbody>
<tr>
<td>H P / Heartstart</td>
<td>Philips</td>
<td>BTE</td>
<td>150 J 150 J 150 J 4 J/kg</td>
</tr>
<tr>
<td>Laerdal/Heartstart</td>
<td>Survival</td>
<td>BTE</td>
<td>150 J 150 J 150 J 4 J/kg</td>
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<tr>
<td>Life-Pak</td>
<td>Medtronic</td>
<td>BTE</td>
<td>200 J 300 J 360 J 4 J/kg</td>
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<tr>
<td>MRL</td>
<td>Welch Allyn</td>
<td>BTE</td>
<td>150 J 200 J 300 J 4 J/kg</td>
</tr>
<tr>
<td>Nihon Kohden</td>
<td>SSEM</td>
<td>BTE</td>
<td>150 J 200 J 270 J 4 J/kg</td>
</tr>
<tr>
<td>Powerheart AED</td>
<td>SSEM</td>
<td>BTE(VE)</td>
<td>200 J 300 J 360 J 4 J/kg</td>
</tr>
<tr>
<td>Responder</td>
<td>Medhold</td>
<td>BTE(VE)</td>
<td>200 J 300 J 360 J 4 J/kg</td>
</tr>
<tr>
<td>Zoll</td>
<td>Stat Medical</td>
<td>Rectilinear</td>
<td>120 J 150 J 200 J 4 J/kg</td>
</tr>
</tbody>
</table>

(BTE = Biphasic Truncated Exponential) (VE = Variable Escalating)

**Labeling of all Defibrillators**

- Labels containing Recommended Energy Settings for Manual Defibrillators are available from the Defibrillator Distributors, and should be placed on each machine (Monophasic and Biphasic). Please contact the appropriate Distributor as soon as possible to ensure that every defibrillator that you may have access to is correctly labeled.

- AED’s that have been modified to comply with the latest international guidelines will be labeled “2006 Protocol Compliant”. AED users are advised to follow the AED voice prompts, and to contact their AED Distributor to upgrade their device as a matter of urgency.

**DEFIBRILLATOR** | **DISTRIBUTOR** | **TELEPHONE** | **FAX** | **CONTACT PERSON**
--- | --- | --- | --- | ---
Hewlett Packard / Heartstart AED | Philips Medical | 011 471 5000 | 011 471 5384 | Renier Hattingh
Laerdal / Heartstart AED | Survival Technology | 011 792 2190 | 011 793 4234 | Janine O’Donnell
Physio-Control / Life-Pak | Medtronic Africa | 011 677 4809 | 011 616 1104 | Tony Soares
MRL | Welch Allyn | 011 777 7555 | 011 777 7556 | Lezanne de Koning
Nihon Kohden / Powerheart AED | Specialised Systems Electro Medical | 011 444 8184 | 011 444 8171 | Lizelle Grindell
Responder | Medhold GEMS | 011 975 0633 | 011 975 3870 | Terence Dobie
Zoll | Stat Medical | 011 462 3112 | 011 462 3113 | Tom Watson

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