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 humanely 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".

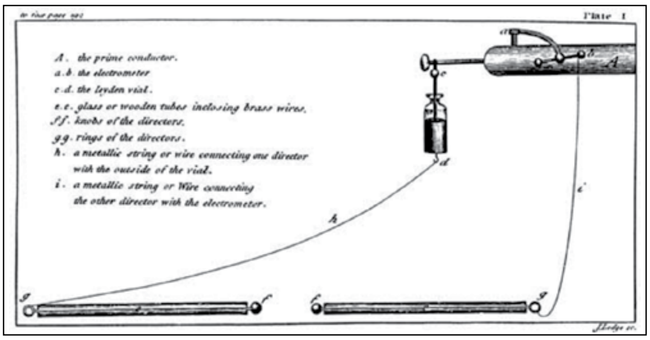

Figure 1: Apparatus as shown in Kite's *An essay on the recovery of the apparently dead* (London, 1788). An electrostatic generator charges a Leyden jar capacitor, which can discharge its accumulated electrical energy through the electrodes below. Energy will build up until the voltage is high enough to jump the spark gap

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.

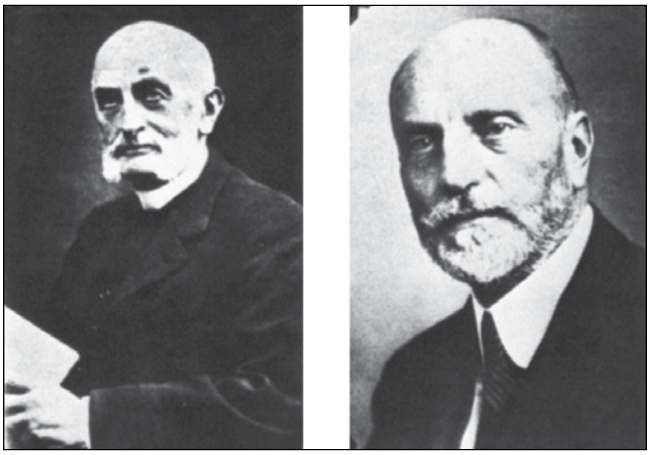

Figure 2a and 2b: Provost (left) and Batelli (right) discovered the impact of electrical stimuli in the production and arrest of fibrillation

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.

Figure 3a: Carl J Wiggers from the Western Reserve University in Cleveland, Ohio

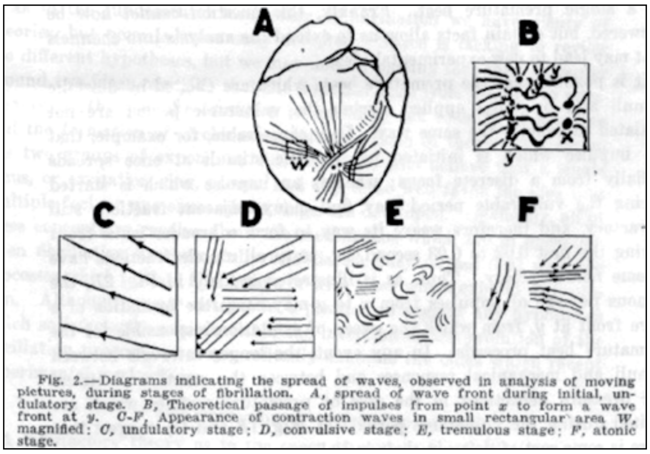

Fig. 2.—Diagrams indicating the spread of waves, observed in analysis of moving pictures, during stages of fibrillation. *A*, spread of wave front during initial, undulatory stage. *B*, Theoretical passage of impulses from point *x* to form a wave front at *y*. *C-F*, Appearance of contraction waves in small rectangular area *W*, magnified: *C*, undulatory stage; *D*, convulsive stage; *E*, tremulous stage; *F*, atonic stage.

Figure 3b: Wiggers' diagram of fibrillation induction, indicating the spread of electrical waves during the various stages of fibrillation

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).

Figure 4: Cardiothoracic surgeon, Claude Beck, in front of the first modern defibrillator that saved a human life

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.

Figure 5: Animal experiments introducing 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 make 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.

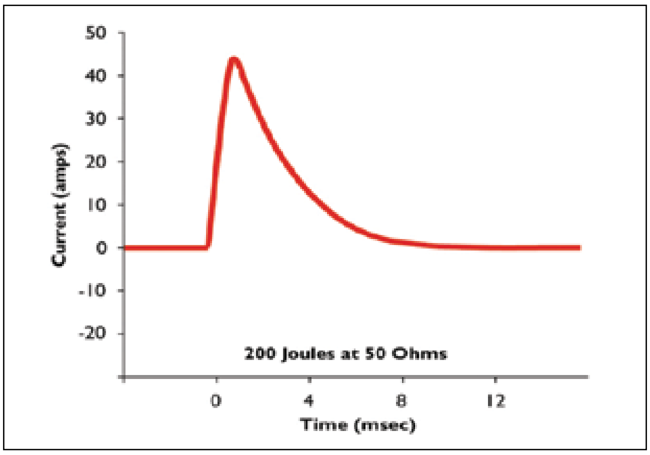

Figure 6: A typical monophasic waveform at 200 J, with current moving in a single direction

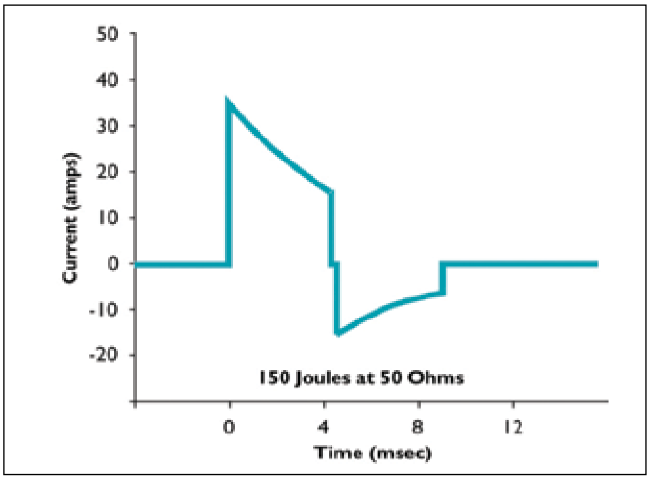

Figure 7: An example of a truncated biphasic exponential waveform showing bidirectional current flow

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 rectilinear biphasic waveform (RBW) was specifically developed for external defibrillation. It sought to improve the earlier biphasics by eliminating high-peak currents and stabilising its shape in the face of varying patient impedance levels. The rectilinear biphasic waveform is characterised by two distinct attributes.

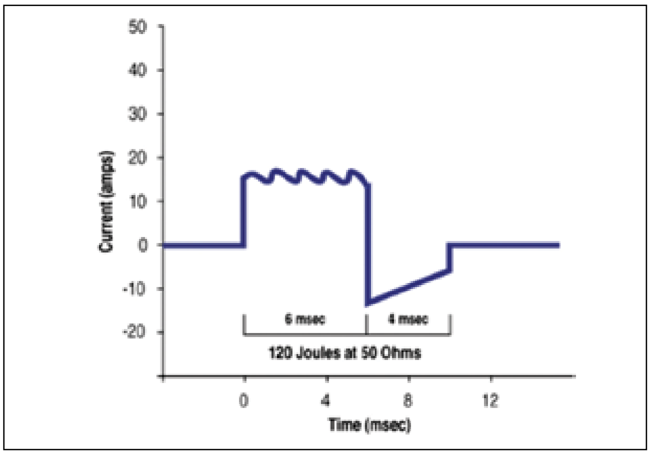

Figure 8: The rectilinear biphasic waveform adjusts for patient impedance during energy delivery

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.

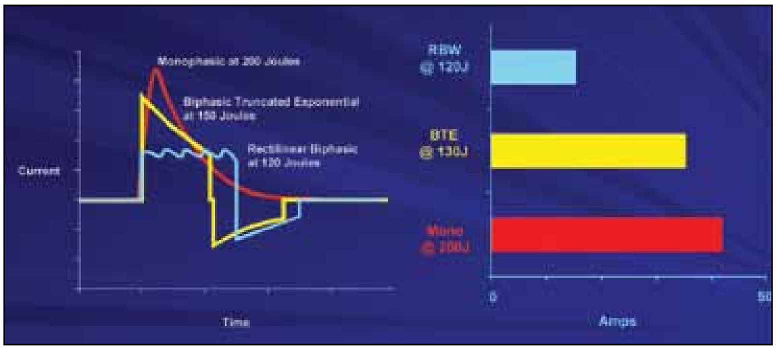

Figure 9: Comparison of monophasic, biphasic truncated exponential and rectilinear biphasic waveforms

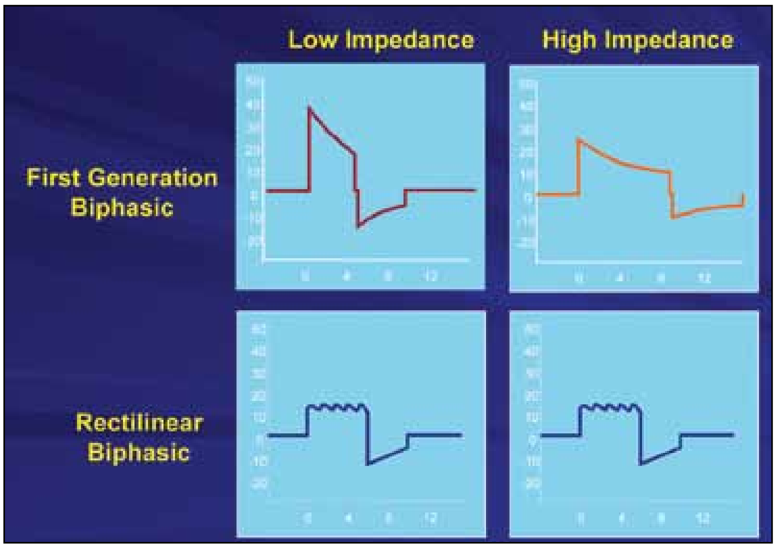

Figure 10: Comparison of various biphasic waveforms, generated when applied to varying levels of patient impedance

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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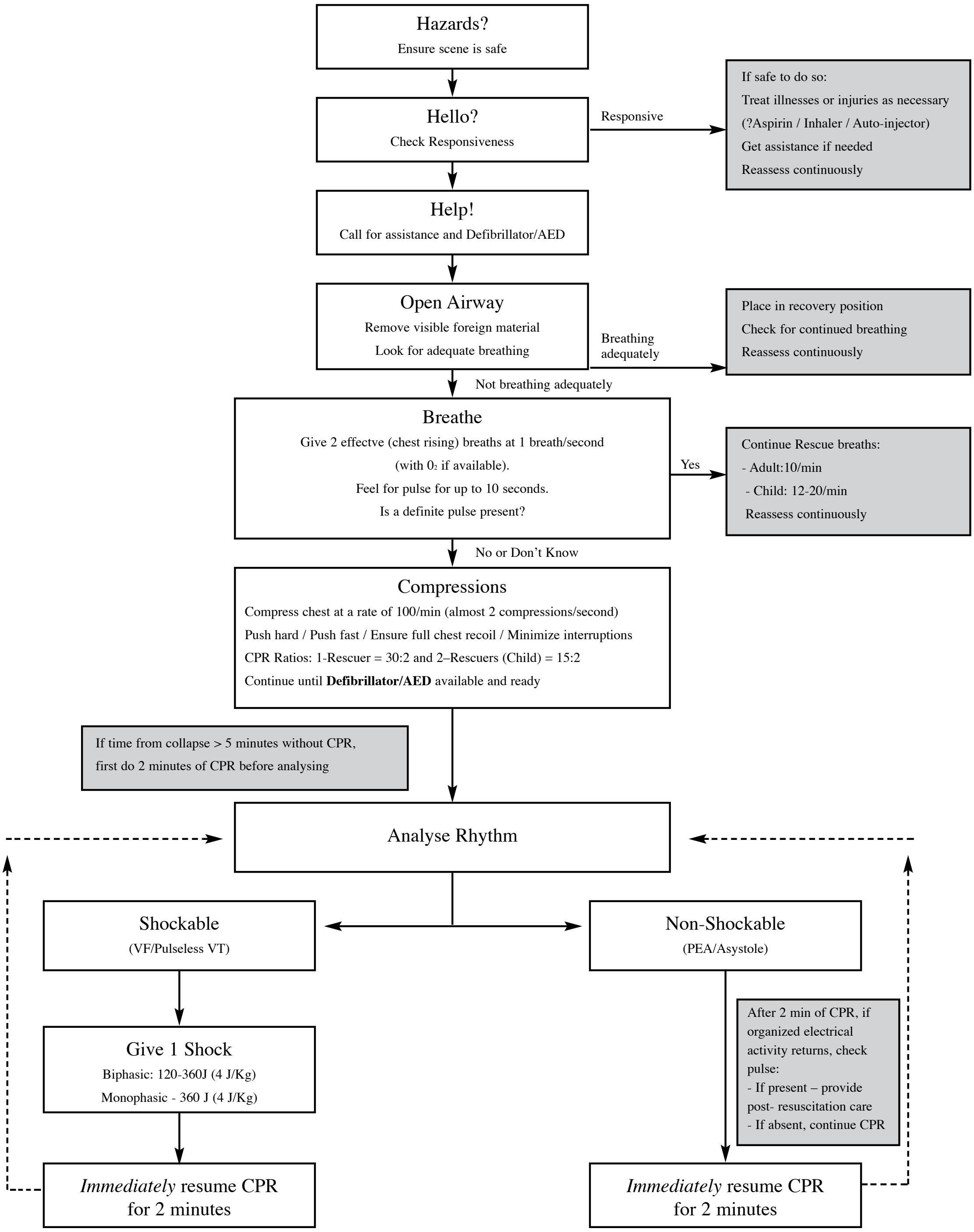
Basic Life Support for Healthcare Providers

(Adult and Child)



Hazards?
Ensure scene is safe

Hello?
Check Responsiveness

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

Help!
Call for assistance and Defibrillator/AED

A

Open Airway
Remove visible foreign material
Look for adequate breathing

Breathing adequately →
Place in recovery position
Check for continued breathing
Reassess continuously

Not breathing adequately

B

Breathe
Give 2 effectve (chest rising) breaths at 1 breath/second
(with 0_2 if available).
Feel for pulse for up to 10 seconds.
Is a definite pulse present?

Yes →
Continue Rescue breaths:
- Adult:10/min
- Child: 12-20/min
Reassess continuously

No or Don’t Know

C

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: 120-360J (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

Immediately resume CPR
for 2 minutes

Do not interrupt chest compressions unless absolutely necessary

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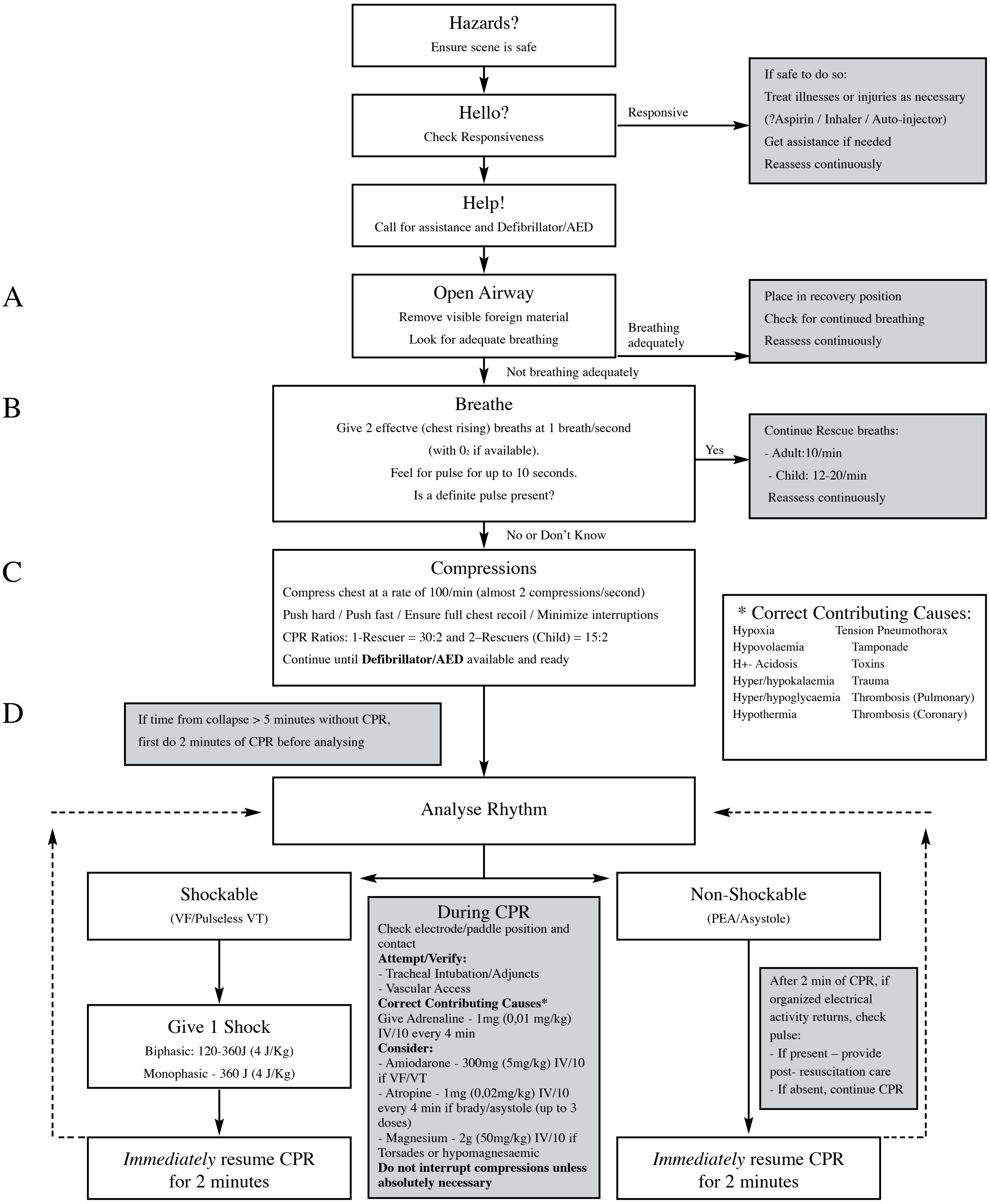
Advanced Life Support for Healthcare Providers

(Adult and Child)



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(with O_2 if available).
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- Adult:10/min
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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

* Correct Contributing Causes:

Hypoxia	Tension Pneumothorax
Hypovolaemia	Tamponade
H+- Acidosis	Toxins
Hyper/hypokalaemia	Trauma
Hyper/hypoglycaemia	Thrombosis (Pulmonary)
Hypothermia	Thrombosis (Coronary)

D

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first do 2 minutes of CPR before analysing

Analyse Rhythm

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

During CPR
Check electrode/paddle position and contact
Attempt/Verify:
- Tracheal Intubation/Adjuncts
- Vascular Access
Correct Contributing Causes*
Give Adrenaline - 1mg (0,01 mg/kg) IV/10 every 4 min
Consider:
- Amiodarone - 300mg (5mg/kg) IV/10 if VF/VT
- Atropine - 1mg (0,02mg/kg) IV/10 every 4 min if brady/asystole (up to 3 doses)
- Magnesium - 2g (50mg/kg) IV/10 if Torsades or hypomagnesaemic
Do not interrupt compressions unless absolutely necessary

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

Immediately resume CPR
for 2 minutes

Do not interrupt chest compressions unless absolutely necessary

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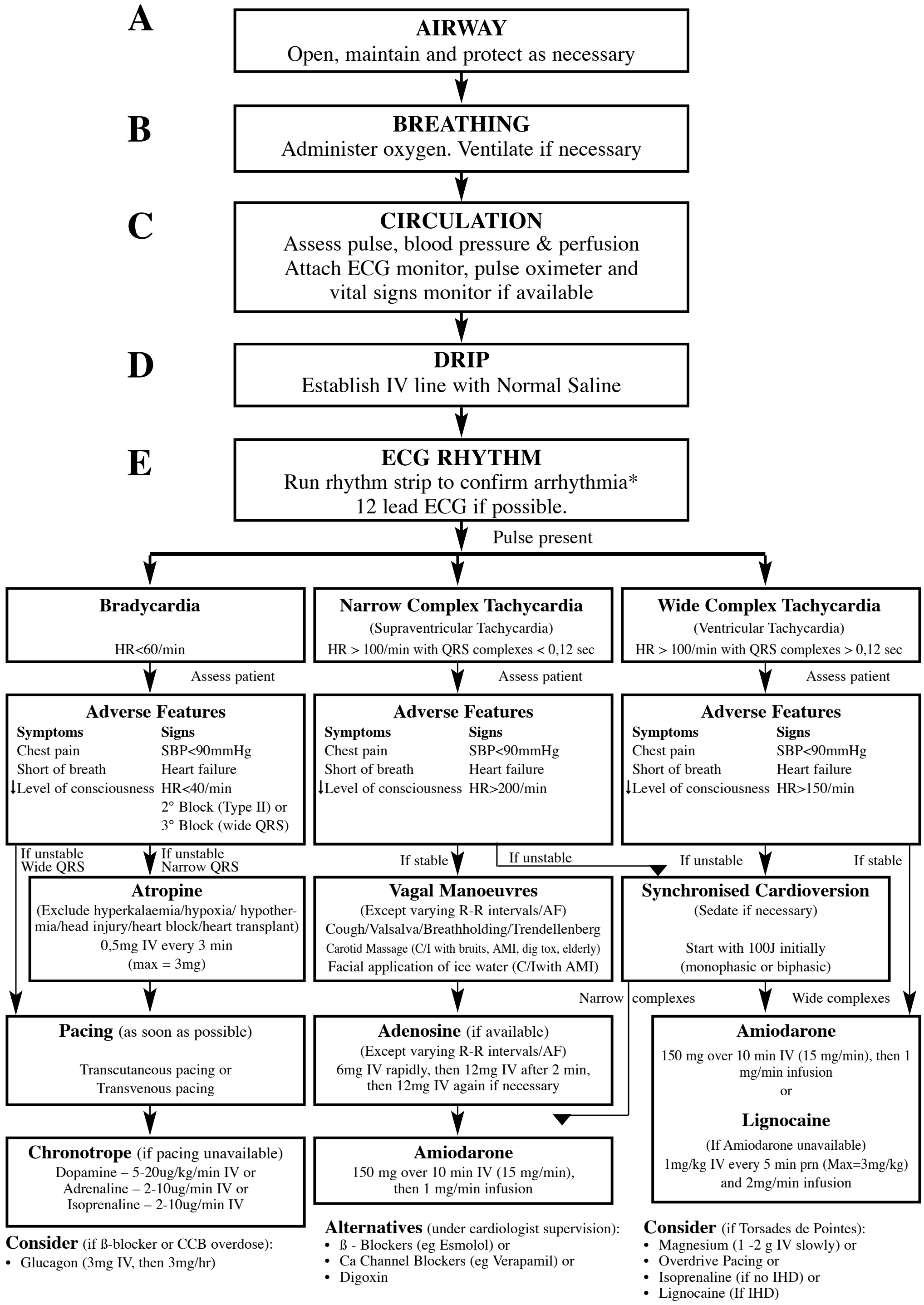
Adult Emergency Arrhythmia Management Algorithm

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A special interest group of South African Heart Association



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.

Pulse present

Bradycardia
HR<60/min

Assess patient

Adverse Features

Symptoms	Signs
Chest pain	SBP<90mmHg
Short of breath	Heart failure
↓Level of consciousness	HR<40/min
	2° Block (Type II) or
	3° Block (wide QRS)

If unstable Wide QRS

If unstable Narrow QRS

Atropine
(Exclude hyperkalaemia/hypoxia/ hypothermia/head injury/heart block/heart transplant)
0,5mg IV every 3 min
(max = 3mg)

Pacing (as soon as possible)
Transcutaneous pacing or
Transvenous pacing

Chronotrope (if pacing unavailable)
Dopamine – 5-20ug/kg/min IV or
Adrenaline – 2-10ug/min IV or
Isoprenaline – 2-10ug/min IV

Consider (if ß-blocker or CCB overdose):
- Glucagon (3mg IV, then 3mg/hr)

Narrow Complex Tachycardia
(Supraventricular Tachycardia)
HR > 100/min with QRS complexes < 0,12 sec

Assess patient

Adverse Features

Symptoms	Signs
Chest pain	SBP<90mmHg
Short of breath	Heart failure
↓Level of consciousness	HR>200/min

If stable

If unstable

Vagal Manoeuvres
(Except varying R-R intervals/AF)
Cough/Valsalva/Breathholding/Trendellenberg
Carotid Massage (C/I with bruits, AMI, dig tox, elderly)
Facial application of ice water (C/Iwith AMI)

Adenosine (if available)
(Except varying R-R intervals/AF)
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):
- ß - Blockers (eg Esmolol) or
- Ca Channel Blockers (eg Verapamil) or
- Digoxin

Wide Complex Tachycardia
(Ventricular Tachycardia)
HR > 100/min with QRS complexes > 0,12 sec

Assess patient

Adverse Features

Symptoms	Signs
Chest pain	SBP<90mmHg
Short of breath	Heart failure
↓Level of consciousness	HR>150/min

If unstable

If stable

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

Narrow complexes

Wide complexes

Amiodarone
150 mg over 10 min IV (15 mg/min), then 1 mg/min infusion
or
Lignocaine
(If Amiodarone unavailable)
1mg/kg IV every 5 min prn (Max=3mg/kg)
and 2mg/min infusion

Consider (if Torsades de Pointes):
- Magnesium (1 -2 g IV slowly) or
- Overdrive Pacing or
- Isoprenaline (if no IHD) or
- Lignocaine (If IHD)

(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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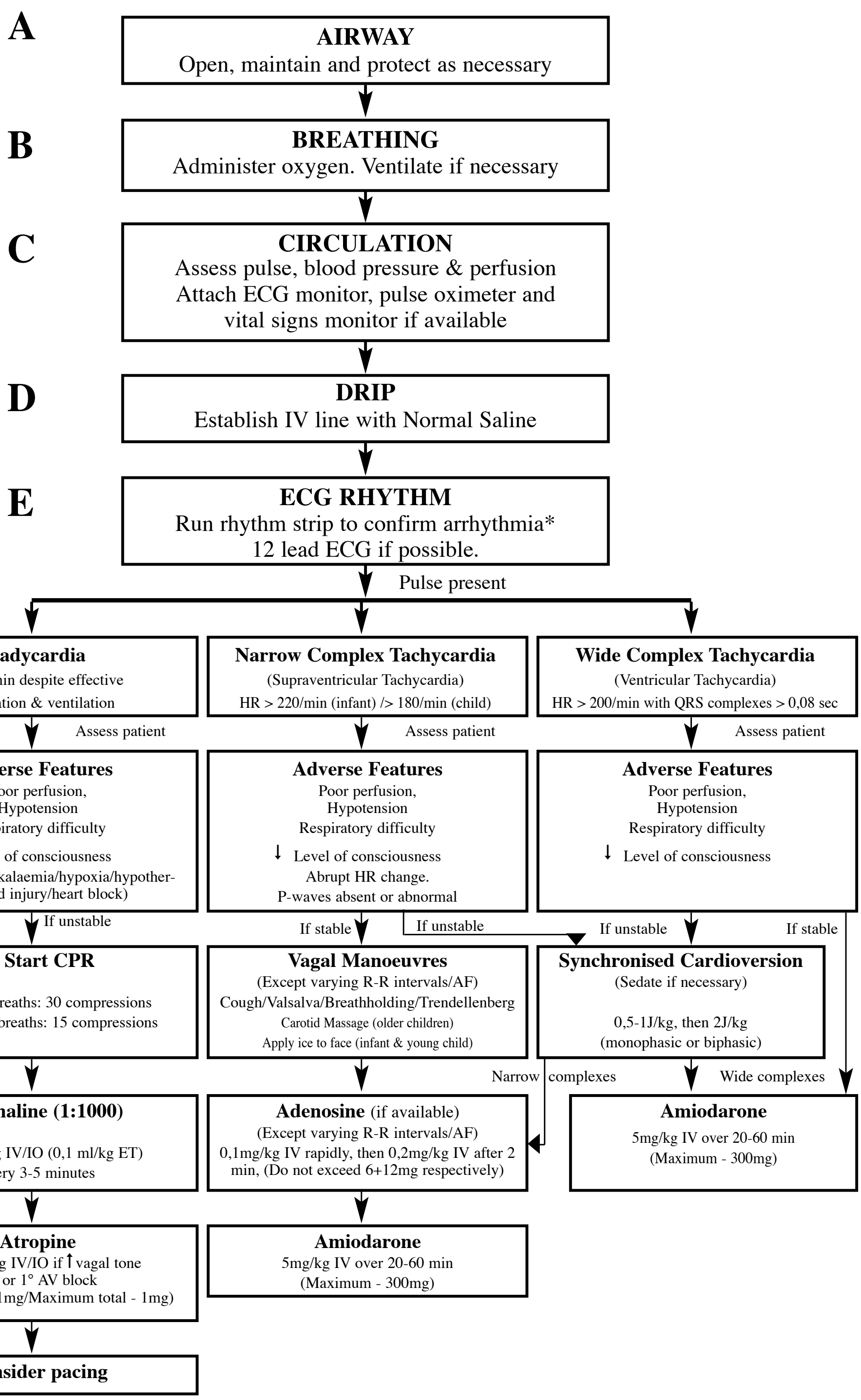
Paediatric Emergency Arrhythmia Management Algorithm



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.

Pulse present

Bradycardia
HR<60/min despite effective
oxygenation & ventilation

Assess patient

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

If unstable

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

Atropine
0,02 mg/kg IV/IO if ↑ vagal tone
or 1° AV block
(Minimum - 0,1mg/Maximum total - 1mg)

Consider pacing

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

Assess patient

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

If stable

If unstable

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

Adenosine (if available)
(Except varying R-R intervals/AF)
0,1mg/kg IV rapidly, then 0,2mg/kg IV after 2 min, (Do not exceed 6+12mg respectively)

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

Wide Complex Tachycardia
(Ventricular Tachycardia)
HR > 200/min with QRS complexes > 0,08 sec

Assess patient

Adverse Features
Poor perfusion,
Hypotension
Respiratory difficulty
↓ Level of consciousness

If unstable

If stable

Synchronised Cardioversion
(Sedate if necessary)
0,5-1J/kg, then 2J/kg
(monophasic or biphasic)

Narrow complexes

Wide complexes

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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2006

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)

Defibrillator Make	Distributor in SA	Biphasic Waveform	Recommended Energy Setting in Cardiac Arrest (Joules)			
			1st Shock	2nd Shock	Subsequent Shock	Paed
H P / Heartstart	Philips	BTE	150 J	150 J	150 J	4 J/kg
Laerdal/Heartstart	Survival	BTE	150 J	150 J	150 J	4 J/kg
Life-Pak	Medtronic	BTE	200 J	300 J	360 J	4 J/kg
MRL	Welch Allyn	BTE	150 J	200 J	300 J	4 J/kg
Nihon Kohden	SSEM	BTE	150 J	200 J	270 J	4 J/kg
Powerheart AED	SSEM	BTE(VE)	200 J	300 J	360 J	4 J/kg
Responder	Medhold	BTE(VE)	200 J	300 J	360 J	4 J/kg
Zoll	Stat Medical	Rectilinear	120 J	150 J	200 J	4 J/kg

(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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