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REVIEW ARTICLE

Anaesthesia for cardioversion

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Summary

Cardioversion is a minor procedure requiring sedation and analgesia. However, it is often performed out-of-hours in remote sites by inexperienced anaesthetists. An understanding is required both of the pathophysiology underlying cardiac arrhythmias and of the technical side of defibrillation equipment, including electrical safety. Patients should have their coagulation status and electrolyte balance checked prior to the procedure to reduce the likelihood of complications. Almost all the available anaesthetic agents have been used for cardioversion in the past, with varying degrees of success. The anaesthetic agent chosen for patients undergoing cardioversion must provide analgesia and sedation, cause the least cardiovascular compromise possible and still enable rapid recovery. Propofol may be the closest anaesthetic agent to this ideal currently available, although careful titration of any agent chosen is also important. Cardioversion may be performed as an emergency, including in the pregnant patient, providing safe anaesthetic practice is followed.

Key words

Cardioversion.
Defibrillation.
Equipment; defibrillator.

Direct current (DC) countershock is a method of converting cardiac arrhythmias back to sinus rhythm. It involves the passage of electric current from an external capacitor-discharge type defibrillator through the heart. It may be used in the emergency situation to terminate life-threatening cardiac arrhythmias or electively to stabilise abnormal rhythms with potential deleterious effects. Cardioversion is the electrical conversion of any rhythm other than ventricular fibrillation and involves synchronisation of the delivered shock with the R wave of the electrocardiograph (ECG).

The earliest recorded successful resuscitation using electricity was performed in 1774 on a 3-year-old near-drowned child using a Leyden jar as the source [1]. The treatment of ventricular fibrillation itself was investigated by Prevost and Batelli, who successfully defibrillated the hearts of animals in 1899 [2]. Alternating current (AC), first used in humans in 1947 [3], was thought to be the definitive method for defibrillation up until the 1960s. However, direct current (DC) shock, which was first used in man in 1962 by Lown [4], was shown to be more effective than alternating current and also produced less skeletal muscle contraction with less risk of causing ventricular fibrillation both in the patient and the operator [5]. Since that time, improvements have been made in the shape of the DC current waveform [6], the timing of shock in relation to the onset of fibrillation [7] and more recently with the advent of automatic external defibrillators [8] and implantable defibrillators [9] capable of automatic rhythm recognition and management.

Patients requiring elective cardioversion need sedation and analgesia as the procedure is painful and distressing [10]. They may have significant pre-existing cardiac disease, including previous myocardial infarction, cardiac failure, angina and hypertension, as well as co-existing diseases of other organ spystems. Attention should be directed to these factors when considering a suitable anaesthetic technique. Patients for cardioversion are often anaesthetised in spare moments between operating theatre lists by junior anaesthetists in unfamiliar surroundings. The urgency of the situation may mean that anaesthesia must be induced in a patient with a full stomach.

This review details the mechanism of action of the defibrillator and the indications for direct current cardioversion. A review of the literature on anaesthesia for cardioversion follows and some guidelines are suggested for safe practice.

Defibrillators, defibrillation and cardioversion

Cardiac physiology

The cardiac rhythm is generated from the conducting system of the heart, consisting of the sino-atrial node, atrioventricular node and the right and left bundles of His.

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Just as the heart responds to an intrinsic electrical impulse from the sino-atrial node, so it will respond to an extrinsic electric impulse. If enough electric current is delivered to the heart, the majority of myocardial cells are depolarised and contract. When the current is removed, if a critical mass of the myocardium is in the recovery phase of the cardiac cycle [11], defibrillation occurs and the sino-atrial node or other intrinsic pacemaker can regain control. This is the underlying principle of defibrillation or cardioversion.

Principles of the defibrillator [5, 12]

The electrical circuitry of a defibrillator is shown in Figure 1. A capacitor is used to store about 160 mC of current, which is discharged over a short period of time; 3 ms is typical. A capacitor consists simply of two plates separated by an insulator. The inductor is included in the circuit to lengthen the current pause.

When a defibrillator discharges, three factors determine the amount of energy delivered to the patient—the potential of the charge, the electrical current and the duration of current.

Stored energy (J) =

Potential difference (V) × Current (A) × Time (s)

and,

Current (A) = Charge (C) per second

Therefore,

Stored energy (J) =

Potential difference (V) × Stored charge (C)

i.e. $E = CV$

Available energy is proportional to the product of the charge and the voltage which decreases from its initial peak. Thus the stored energy is, in fact, only half that calculated from the initial peak potential.

For 5000 V potential, 160 mC of charge is produced, thus at maximum output:

$$\text{Energy} = \tfrac{1}{2} \times 160\ \text{mC} \times 5000\ \text{V} = 400\ \text{J}$$

There is a further complicating factor. The energy stored by the defibrillator is not the same as the energy delivered to the patient. The latter depends upon the transthoracic impedence of the patient which is typically 50 ohms (Ω),

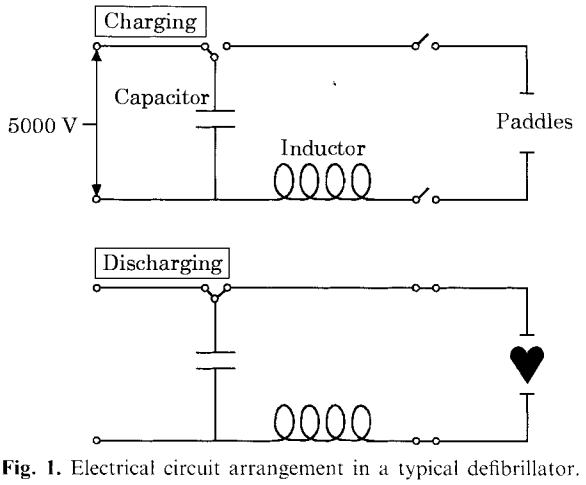

Fig. 1. Electrical circuit arrangement in a typical defibrillator.

although this may vary depending on the inter-electrode distance, electrode-chest coupling medium (i.e. the gel), previous shocks, skin resistance, sweating and the phase of ventilation of the patient's lungs [13, 14]. When defibrillators are tested, a test impedance of 50 Ω is used. Some defibrillators indicate the actual energy delivered to the patient following discharge.

The transthoracic impedance is standardised as much as possible between patients by using large paddles, applying conductive electrode gel to the skin and applying firm pressure. It is important to use the correct paddle position during cardioversion [15]. Paddles should not be placed over the sternum because bone is a poor conductor, but should be placed antero-posteriorly or antero-laterally. No difference in likelihood of success has been shown between these two routes [16]. Paddle size is also important; large paddles reduce the impedance to current flow and reduce the possibility of myocardial damage. However, there is also a maximum size, to avoid the possibility of the paddles touching and to ensure that all the current passes through the heart [17].

The success of cardioversion has also been shown to be related to the phase of ventilation in which it is carried out. Electrical impedance is at its lowest when the thoracic volume is at a minimum. Thus cardioversion should ideally be carried out during the expiratory phase of ventilation [13].

Electrical energy requirements

In rhythms affecting the whole myocardium—ventricular tachycardia, for instance—a large current is used, starting with 200 J. In atrial flutter or fibrillation, however, only the atria are affected. The current required is thus reduced and only 25–100 J may be necessary for effective cardioversion [18]. It has also been shown that successive shocks actually reduce the transthoracic impedance by a significant amount, up to 8% for the first shock [13]. This is a further reason why a second or subsequent shock may be successful when the first has failed.

Much smaller quanta of energy are required during internal defibrillation which is performed when the chest is open, for example during cardiac surgery. In this case, the high electrical impedance of the lungs is removed and the optimum initial energy for internal defibrillation is only 10 to 20 J [19]. Larger shocks than this can produce myocardial necrosis. Paddles, handles and cables must, of course, be sterilised or autoclaved before use.

Electrical safety

Defibrillators must conform to Type CF medical electrical equipment (labelled with the symbol ♥). This means that they are class I or II mains powered equipment with maximum patient leakage current of 10 μA in normal condition and 50 μA with a single-fault connection [20]. Class I equipment is earthed, class II is doubly-insulated and electrically isolated from ground.

Indications for cardioversion [15, 17, 21] (Table 1)

There is a clear distinction between elective and emergency cardioversion. Emergency cardioversion (called defibrillation if the underlying rhythm is ventricular fibrillation) is

Table 1. Indications for elective cardioversion [15, 17, 21].

Ventricular tachycardia
Atrial flutter
Arrhythmias due to Wolff-Parkinson-White syndrome (atrial tachycardia, flutter or fibrillation)
Atrial fibrillation
1. Onset < 1 year
2. History of systemic embolism
3. Persistent after treated thyrotoxicosis or cardiac surgery
4. Fast ventricular response, not controlled by drugs
Supraventricular tachycardia when other therapy has failed

commonly required for ventricular arrhythmias, although any cardiac arrhythmia which causes acute cardiovascular compromise, such as atrial fibrillation with a fast atrio-ventricular conduction rate, may require urgent cardioversion. Under these circumstances, the patient may have lost consciousness because of the poor cardiac output and anaesthesia or analgesia may not be necessary or desirable.

Patients with limited cardiovascular compromise, but with rhythms which are unstable or which predispose to complications such as thrombus formation, require cardioversion, but in these cases it may be undertaken as an elective procedure. In practice, most of these rhythms are supraventricular in origin. The commonest indication for elective cardioversion is atrial fibrillation and cardioversion is successful in up to 90% of these patients although there is a high incidence of relapse [14, 22]. The length of time that the patient has been in atrial fibrillation is the most important factor in determining the likelihood of success of the procedure [14]. There are no absolute contraindications to cardioversion, although Table 2 lists some of the relative contraindications.

Patients undergoing elective cardioversion require an anaesthetic as they are conscious and the procedure is painful [10]. These cases may be delayed until the patient is fasted, thus removing some of the anaesthetic problems.

Synchronisation

Following cardiac contraction, there is a period of unresponsiveness to electrical stimulation called the refractory period. This may be further divided into the absolute refractory period, when no amount of stimulation can cause depolarisation and the relative refractory period, when supramaximal stimuli can cause depolarisation. The ventricular refractory period is represented by the T wave of the ECG. Depolarisation of the heart during the relative refractory period is associated with a high incidence of ventricular fibrillation. For this reason, this period of the cardiac cycle is known as the vulnerable period [15] (Fig. 2). The risk of provoking ventricular fibrillation using a non-synchronised discharge is still less than 5%.

Table 2. Relative contraindications to elective cardioversion.

Atrioventricular block
Beta adrenoceptor blockade
Abnormal clinical biochemical results – particularly hypokalaemia
Digoxin toxicity
Poorly controlled anticoagulation
Atrial fibrillation with a slow ventricular response rate

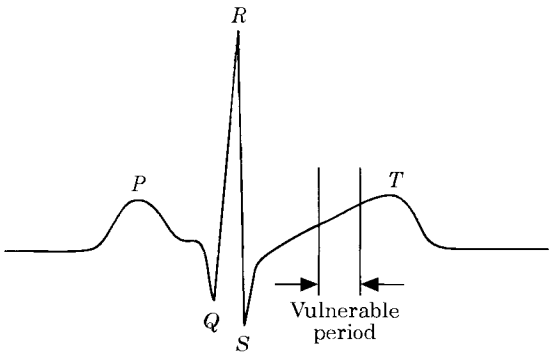

Fig. 2. The 'vulnerable period' of the ECG.

During cardioversion, it is clearly important to avoid precipitation of a more unstable rhythm than that already present. Thus for elective cardioversion, with a rhythm in which there are relatively normal QRS complexes on the ECG, the technique of synchronised DC shock is used to minimise this risk. The defibrillator 'marks' an R wave by using criteria such as its steep slope and relatively large amplitude. When the defibrillator discharge button is pressed, the shock is not delivered until the next R wave is recognised by the machine.

Anaesthesia for cardioversion

Patient preparation

Cardioversion is frequently performed in areas where anaesthesia is rarely used, such as the coronary care unit. For this reason, it is essential that adequate staff, equipment and preparation are available for the procedure. This means a trained operating department assistant or anaesthetic nurse, an anaesthetic machine, an oxygen source, suction apparatus, a means of providing intermittent positive pressure ventilation as well as an anaesthetist with experience of cardioversion [23]. The anaesthetic machine and other equipment should be checked before use. Standard monitoring as recommended by the Association of Anaesthetists of Great Britain and Ireland should also be employed [24].

Some patients undergoing cardioversion will be anticoagulated to prevent systemic embolisation [25]. It is important to check that the degree of anticoagulation is adequate prior to the procedure. Similarly, current serum electrolyte values should be checked. Patients who are receiving glyceryl trinitrate patches for angina or cardiac failure should have these removed prior to cardioversion as there is a risk of an explosion with electric sparks.

It would be unusual to premedicate patients for cardioversion routinely, although some authors have recommended the use of anticholinergic drugs such as atropine given immediately prior to anaesthesia to avoid the precipitation of vagotonic bradyarrhythmias during the procedure [26].

Choice of induction agent

The ideal anaesthetic agent for use in patients undergoing cardioversion should produce rapid loss of consciousness

with some analgesia, rapid recovery and should not accumulate on repeated dosing. It should be devoid of cardiovascular side-effects and in particular should have minimal effects on myocardial contractility and conduction. It should not cause vomiting or other side effects and should have a short elimination half-life, which is not prolonged in patients with renal or hepatic impairment.

None of the anaesthetic agents currently available possess all of these qualities. However, all the intravenous induction and sedative agents have been tested, with various degrees of success. The first sedative agents used were benzodiazepines, chosen because of their amnesic and sedative properties. Diazepam was the original drug used for sedation [27–29], having been shown to be relatively cardiovascularly stable in poor risk patients [30] and was associated with a lower incidence of ventricular extrasystoles than thipentone [31]. However, in one study, one third of patients given diazepam were not amnesic for the procedure [32]. Diazepam has been largely superseded by midazolam, which has a shorter duration of action, produces less pain on injection and is associated with a lower incidence of allergic response [33, 34]. Midazolam has been used with the benzodiazepine antagonist flumazenil to prevent residual sedation [35], although one study showed prolonged sedation with midazolam even after the patients had been given flumazenil [36]. Flunitrazepam has also been used [37] although there appears to be no particular reason why it should offer any advantage over midazolam.

The barbiturates are another group of drugs used frequently for anaesthesia for short procedures. Thiopentone was, in fact, the sedative drug used by Lown in his original case report of DC cardioversion in humans [38]. Both thiopentone [36, 39–42] and methohexitone [43] have been successfully used for anaesthesia for cardioversion, producing rapid anaesthesia and recovery. Disadvantages of the barbiturates include the need for pre-mixing [43]; a greater degree of apnoea [32, 36]; being subjectively less pleasant than propofol [41]; and a reduction in systolic arterial pressure. Lignocaine has been given with thiopentone in an attempt to reduce its cardiovascular depressant effects [44]. One other major disadvantage of the barbiturates is that they accumulate when given in repeated doses, so if the procedure is prolonged for any reason, second and subsequent doses may cause recovery to be prolonged.

Etomidate is traditionally held to be an appropriate agent to use for patients with cardiovascular compromise, being cardiovascularly stable with a low incidence of allergic side effects. However, many anaesthetists avoid using it because of adverse effects on adrenal cortical steroid biosynthesis first highlighted in 1984 [45]. Etomidate has been used alone for cardioversion [39, 40, 46, 47], or with fentanyl to provide analgesia [48]. However, it produces pain on injection in most patients, prolongs recovery compared with propofol [49], causes myoclonus on induction which may interfere with electrocardiographic interpretation [39, 46, 47, 49] and causes postoperative nausea and vomiting. These complications are in addition to the inhibitory effects of etomidate on the adrenal cortex, which may occur even after a single dose [50].

Propofol is probably the drug which most closely approaches the ideal agent for cardioversion [36, 41, 47, 51, 52]. It is formulated as a ready-to-use solution, is subjectively pleasant at induction, produces rapid loss of consciousness and amnesia and may be given in repeated doses if the procedure takes longer than expected, without accumulation and prolongation of recovery. The incidence of pain on injection of propofol is low if it is pre-mixed with small amounts of lignocaine. The only major drawback of propofol is the decrease in blood pressure which accompanies bolus administration. This has been successfully combated by giving it as an infusion rather than by bolus injection [47].

Historically, other intravenous anaesthetic agents such as Althesin [53] and propanidid [32] were used for anaesthesia for cardioversion in the past but have been withdrawn from use. Eltanolone (pregnanolone) is a new steroid-based anaesthetic agent which has some of the features of the ideal induction agent and may be suitable for cardioversion [54].

Pre-oxygenation

Pre-oxygenation for a period of several minutes is recommended for all patients undergoing cardioversion, not just those requiring rapid sequence induction. It provides a margin of safety if there are subsequent unexpected problems in ventilation or oxygenation and reduces the incidence of desaturation episodes in spontaneously-breathing patients following induction of anaesthesia [55, 56].

Cardioversion for the patient with a full stomach

If cardioversion is required as a matter of extreme urgency it may be necessary to induce anaesthesia in a patient with a full stomach. Anaesthetic considerations regarding this outweigh considerations of convenience or unavailability of equipment. Measures must be taken to secure the airway against regurgitation and aspiration of gastric contents. This will involve pre-oxygenation, tracheal intubation and the use of depolarising muscle relaxants. Suxamethonium is not contraindicated in these patients, but bradyarrhythmias may be precipitated and hyperkalaemia exacerbated. The serum potassium level should be within normal limits.

Cardiovascular stability

Whichever induction agent is chosen, careful titration of the drug should be used to avoid overdose. Some consideration should be given to the depth of anaesthesia required. The procedure is stimulating and painful, therefore sufficient anaesthetic depth must be reached to avoid a sympathetic hypertensive response which could be harmful. Suitable end points for induction are loss of verbal contact or eyelash reflex.

The most dangerous side effect of the anaesthetic induction agents in cardioversion is their ability to cause depression of an already dysfunctional cardiovascular system. Etomidate may be the safest agent in this respect, but it has other deleterious effects. Infusions of anaesthetic drugs have been used and shown to produce less cardiovascular depression than bolus doses [47].

Recovery

After restoration of normal sinus rhythm, rapid return to full consciousness with recovery of normal airway reflexes is desirable. Patients undergoing cardioversion are rarely adjacent to the usual recovery areas and are frequently

recovered in the place where they were anaesthetised. Despite this, facilities for suction and oxygen administration and a full range of emergency drugs should be immediately available. A trained nurse must stay with the patient until recovery is complete.

Cardioversion in pregnancy

This has been reported by several authors [57–59] and has been shown to be safe for the mother and baby, although it has led to fetal arrhythmias [60]. These cases present the combined problems of anaesthesia in a pregnant woman and the use of anaesthetic drugs in a patient with a compromised circulation.

Complications of cardioversion

Most anaesthetic complications following elective cardioversion are related to cardiovascular depression. Airway complications are rare since tracheal intubation and the use of suxamethonium are uncommon. However, any anaesthetic complication may follow anaesthesia for cardioversion. Many other complications have been described, including ventricular fibrillation [61], death due to synchronisation failure of a defibrillator [62], elevation of cardiac enzyme levels [63] (although this is mainly from skeletal muscle damage [64]), rhabdomyolysis and renal failure [65], systemic embolisation [66, 67] and severe bradycardia [68]. It is advisable for the anaesthetist to remove the oxygen source from the patient prior to cardioversion or defibrillation as there have been reports of burns occurring to patients when oxygen has been left running in the close vicinity of the patient. A hazard warning was issued concerning this [69].

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