Complications of Direct Current Cardioversion

including sedation-related complications and comparison with pharmacological cardioversion

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# Introduction

Direct current electrical cardioversion is the preferred method to terminate acute atrial fibrillation in the United States <looking for reference to document this -please help>. This preference appears to be based on the impression that it is safer, faster and more effective than pharmacological conversion. The primary purpose of this report is to examine adverse effects of electrical cardioversion, including those related to sedation. Overall advantages and disadvantages of the electrical and pharmacological cardioversion will be discussed.

# Complications of Electrical Cardioversion

Known complications of electrical cardioversion are listed in Table 1 in rough order of medical severity. Reliable incidence rates for these adverse effects are not uniformly available. Each complication is discussed below. Thromboembolic and bradycardia events are not included, as they are expected to be similar for the electrical and pharmacological methods.

Table 1: Complications of Electrical Cardioversion

1. Arrhythmia induction
2. Rhabdomyolysis
3. Renal failure
4. Pulmonary edema
5. Myocardial injury
6. Skeletal muscle injury
7. Skin burn

## Arrhythmia Induction

Ventricular tachyarrhythmias may result from attempted electrical cardioversion, and may result in death[1, 2]. It is, however, a rare complication. In a study of 760 patients undergoing cardioversion for AF, there was only one episode of ventricular tachycardia (and cardiac arrest) – 0.13%[3]. Causes for this complication include poor synchronization and low shock energy[4, 5].

## Rhabdomyolysis

There are several reports of rhabdomyolysis occurring after electrical cardioversion[6-8]. It is a rare complication, though the incidence rate is unknown. It appears to be related to skeletal muscle injury, usually in association with multiple countershocks, resulting myoglobinuric renal failure.

## Renal Failure

Renal failure unrelated to myoglobinuria may occur after electrical cardioversion[9-11]. Hellman and colleagues[9] reported a rise in serum creatinine of 25% or more in 17% of 112 consecutive patients undergoing cardioversion for AF. Presence of heart failure was a risk factor for this complication. One of the patients required hemodialysis. Extent of recovery or progression of renal dysfunction was not examined in this study. The study of Schmidt and colleagues[11] showed a return to the group average pre-cardioversion eGFR at 12 months of follow-up. Mechanisms responsible for renal impairment electrical after cardioversion are unclear.

## Pulmonary Edema

Pulmonary edema is a known adverse effect of electrical cardioversion[2, 9, 12, 13]. Gowda, et al[12] reported on 30 subjects in a meta-analysis of available literature. Most events occurred early after cardioversion (52%). Potential mechanisms are dyssynchrony in atrial recovery (right sooner than left) and transient shock-related left ventricular dysfunction[14]. Gowda[12] did not report on the incidence rate, which is unknown.

## Myocardial Injury

There is extensive evidence in the literature for myocardial stunning injury and resultant cardiac dysfunction following electrical cardioversion[14-22]. In the early animal study of Ehsani, et al[17], 11 dogs receiving ten 240 joule countershocks, CPK was elevated in all, and in 6 dogs, MB CPK was elevated, electrocardiographic ST elevation was present, and myocardial necrosis was identified histologically. In another early study in dogs, Wilson and colleagues[22] found extensive ST elevation and macroscopic histological abnormalities in dogs receiving 5 or more shocks with a variety of waveforms. In a clinical component of their study, Ehsani[17] reported that 15 of 30 patients undergoing a single DC cardioversion had CPK elevations, and 2 had abnormal MB CPK levels. Stabilini et al[20] reported increased MB CPK in 2 of 6 patients undergoing electrical cardioversion. Chun and colleagues[16] reported transient ST elevation in 3 of 3 electrically cardioverted patients. Cassin, et al[15], reported that 6 of 142 electrically cardioverted patients had marked ST elevation. In a larger study of 64 patients undergoing electrical cardioversion for a variety of arrhythmias, post-conversion ST elevation was observed in 11 (17%) and ST depression in 25 (39%)[18].

In some studies, however, in the presence of CPK and MB CPK elevations after cardioversion, troponin levels were not elevated in patients receiving single shocks, suggesting that the abnormal enzyme release was from skeletal tissue[19].

Apparent myocardial stunning after electrical cardioversion has been reported in a patient[21], leading to death. Kobayashi and coworkers[14] demonstrated echocardiographically the development of left ventricular diastolic dysfunction in a patient who went into pulmonary edema after cardioversion. However, there are no controlled experiments of which we are aware describing the effect of electrical countershocks on LV performance in animals or humans in sinus rhythm. Reports exist on effects of cardioversion of sustained tachyarrhythmias on hemodynamics in animals and humans, but, as expected, these reports show hemodynamic improvement with restoration of sinus rhythm, but do not help us evaluate the independent effect of countershocks on intrinsic hemodynamics.

Overall, it seems likely that electrical cardioversion can adversely affect the myocardium. However, the extent of this effect in humans undergoing a single shock is not clear.

## Skeletal Muscle Injury

Several of the citations above[15-17, 20] demonstrate substantial skeletal muscle enzyme leak after electrical cardioversion, and the reported cases of rhabdomyolysis[6-8] further substantiate this adverse effect, and there is considerable additional evidence for skeletal muscle damage after electrical cardioversion[23-28]. Except in the few cases of rhabdomyolysis, it appears that muscle injury is rarely symptomatic or clinically consequential in patients receiving single countershocks.

## Skin Burns

Skin burns related to external electrical cardioversion are commonly reported [2, 29-38]. Ambler and colleagues[30] comprehensively evaluated skin burns after elective electrical cardioversion. In 83 subjects receiving from 1 to 8 countershocks. Shock strength was below 600 joules in 60 (72%). Two hours after cardioversion the average erythema index was higher in paddle locations compared with other sites, and skin temperature was also higher. Sensory detection was significantly reduced at paddle sites. At 2 or 24 hours 84% of patients experienced pain at paddle sites. The pain was moderate or severe in 23%. The occurrence of pain was related to the total energy and number of shocks received. A number of procedural and equipment modifications have been recommended for mitigation of skin burns, but none are completely effective[29, 32, 34].

# Complications of Sedation

A list of known complications of sedation can be found in Table 2. Most of the information is derived from sedation for procedures other than electrical cardioversion, and those procedures are often longer in duration the cardioversion, requiring more prolonged sedation. The overall incidence of significant complications is about 0.8%, and somewhat higher for deep compared with moderate sedation (0.9% vs. 0.5%)[39].

## Death

Table 2: Complications of Sedation

1. Death
2. Desaturation
	1. Hypoventilation
	2. Airway obstruction
3. Aspiration
4. Nausea and vomiting
5. Hypotension
6. Paradoxical excitement
7. Cognitive impairment

Death is a known complication of sedation for procedures. Though uncommon, there are several reports on this complication[40-48]. Most are caused by cardiorespiratory problems, including aspiration. Its true incidence is not known. The most popular sedative, propofol, has been implicated in sedation-related deaths, but these cases may be isolated to prolonged infusions[49-52].

## Desaturation

Desaturation during sedation is common[53-62], and may result primarily from (in order of estimated frequency[39]) laryngospasm, airway closure, hypoventilation, bronchospasm, or aspiration. The incidence of severe desaturation (defined as O2 saturation <88%) has been reported to be 0.12% during sedation for GI endoscopy[62], and 4% when defined as O2 saturation <90%[54]. In a study evaluating use of capnography to reduce desaturation events during propofol sedation for colonoscopy[53], desaturation below 90% occurred in 19.8% and below 85% in 7.8%. These event rates were improved with capnography monitoring. Generally, desaturation can be reversed by interruption of the infusion or by positive pressure ventilation, and ameliorated by oxygen supplementation.

## Aspiration

Aspiration during GI endoscopy is reported to be “fairly common”[48]. It is often signaled by coughing, and its reported incidence is quite high (13%)[54]. In the same study the incidence of coughing 3 or more times, which might be more suggestive of aspiration, was 9%. Five or more coughing events occurred in 4%. Aspiration can lead to desaturation, and, in some cases, pneumonia. Kollman and colleagues reported an incidence of 2.6% in a case-control study of 250 in patients of 65 years or older[63]. In that same group the incidence of a systemic inflammatory response after endoscopy was 7.8%.

## Nausea and Vomiting

Nausea and vomiting are well known adverse effects of opioids, which are responsible for most sedation-related nausea and vomiting. In a retrospective study of 1,180 general anesthesia or intravenous sedation[64], the incidence of nausea and vomiting was 6% in the sedation group (and 14% in the general anesthesia group). This complication can be ameliorated with antiemetics. Its most serious outcome is aspiration and its complication.

## Hypotension

Sedation commonly reduces blood pressure. In 757 patients undergoing non-emergent GI endoscopy, the average decrease in blood pressure was 11.1 mmHg (-7.3%) for systolic and 6.1 mmHg (-5.6%) for diastolic. As reported, “the maximum drop in SBP was <10% in 28% of patients, 10–19% in 26% of patients, 20–29% in 25% of patients, 30–39% in 14% of patients, 40–49% in 6% of patients, and >50% in 1% of patients.” Systolic pressure below 90 mmHg was observed in 23.9% of patients. While in this study, there were no serious sequelae of hypotension, a fall in systolic pressure of 30% or more, which was seen in 21%, or a decrease below 90 mmHg, which occurred in 23.9%, could have deleterious effects in vulnerable patients with cardiovascular impairment.

## Paradoxical excitement

Paradoxical excitement (disinhibition) is a well-known adverse response to benzodiazepams, propofol and other sedatives. It consists of agitation and body movement. The incidence rate of severe disinhibition in patients undergoing endoscopic procedures is reported to be 1.4% for midazolam[65] and 2.6% for propofol[66]. History of alcohol abuse greatly exacerbates the risk for propofol[66]. Disinhibition can be severe enough to force cancellation of the procedure.

## Cognitive impairment

Procedural sedation is expected to completely impair cognition during the procedure, but impairment persists for a period of time after consciousness returns. Padmanabhan and colleagues studies the relative effects of propofol alone and in combination with midazolam on post-procedural cognitive impairment[67]. They found that the effects of propofol alone and in combination were similar. Patients were interviewed before they were discharged from the hospital in accordance with hospital criteria (which did not include formal cognitive testing. One hundred patients were included in the two treatment groups. Cognitive assessment tests revealed significant cognitive impairment at the time of hospital discharge in 18.5% of patients. The average post-procedure time of discharge was 65 minutes.

# Relative Advantages and Disadvantages of Pharmacological and Electrical Cardioversion

Advantages and disadvantages of each method are summarized in the four tables below.

Table 3: Advantages of Electrical Cardioversion

1. High efficacy
2. No adverse drug effects

Table 4: Advantages of Pharmacological Cardioversion

1. Faster -no coordination or scheduling
2. No sedation complications
3. None of the other DCCV adverse effects
4. Lower cost

Table 5: Disadvantages of Electrical Cardioversion

1. Sedation
2. Slower
	1. Coordination with anesthesiology
	2. Scheduling around meal status
3. Higher cost

Table 6: Disadvantages of Pharmacological Cardioversion

1. Proarrhythmic drug effects
2. Negative inotropic drug effects
3. Lower efficacy

While electrical cardioversion is generally more frequently successful and is not associated with antiarrhythmic drug side effects, conversion will usually be achieved faster by pharmacological intervention because delay due to a recent meal and coordination with anesthesiology are not necessary. Pharmacological intervention is usually associated with a lower cost because costs related to anesthesiology are not incurred, and the adverse effects of sedation are obviated. Though electrical cardioversion seems to be the preferred method to convert acute AF in the United States, this preference may not be justified if a convenient-to-use, highly effective drug with minimal proarrhythmic adverse effect were available.

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