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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Haemodynamic effects of etomidate, propofol and electrical shock in patients undergoing implantable cardioverter-defibrillator testing

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Abstract

Background: Anaesthetic drugs and internal electrical shock may alter the haemodynamic status of patients undergoing implantable cardioverter-defibrillator (ICD) testing. Comparative data on the mechanisms of etomidate and propofol-induced changes in haemodynamic parameters are inconsistent. Also the effects of ICD shock on haemodynamics have not been extensively studied.

Aim: To compare the haemodynamic effects of etomidate and propofol as well as electrical shock during ICD testing in a prospective, randomised trial.

Methods: The study group consisted of 63 consecutive patients (mean age 66 ± 10 years, 51 males) who underwent ICD testing. Haemodynamic parameters were measured using impedance cardiography (Task Force Monitor Systems, CNSystems, Austria) before and after injection of etomidate (n = 30) or propofol (n = 33) as well as immediately after internal defibrillation of ventricular fibrillation (VF). Parameters measured included heart rate, systolic (sBP), diastolic (dBP) and mean (mBP) blood pressure, stroke volume (SV), cardiac output (CO) and total peripheral resistance (TPR).

Results: Propofol significantly decreased the values of all measured parameters (sBP: 123.4 ± 17.1 vs. 106.3 ± 18 mm Hg, $p < 0.0001$; dBP: 83.7 ± 12.2 vs. 74.1 ± 13.8 mm Hg, $p < 0.0001$; mBP: 93.9 ± 13.1 vs. 81.1 ± 16.1 mm Hg, $p < 0.0001$; SV: 61.1 ± 19.3 vs. 56.4 ± 15.7 mL, $p < 0.003$; CO: 4.51 ± 1.07 vs. 4.17 ± 0.73 L/min, $p < 0.003$; and TPR: 1,735.8 ± 532.6 vs. 1,573.9 ± 390.5 dyn×s/cm^5), whereas the only significant change following etomidate infusion was a decrease in SV (60.6 ± 11 vs. 56.8 ± 10 mL, $p < 0.022$). The propofol-induced changes were similar in patients with reduced (< 40%) vs. preserved (≥ 40%) left ventricular ejection fraction (LVEF) and in patients in heart failure NYHA class 0–II vs. class III–IV. Induction of VF and internal electrical shock did not cause major haemodynamic changes apart from significant, albeit very modest, drops in dBP and mBP (77 ± 2 vs. 72.9 ± 18 mm Hg, $p < 0.002$, and 85.2 ± 17 vs. 81.8 ± 20 mm Hg, $p < 0.017$, respectively). There were no complications during ICD testing.

Conclusions: Propofol significantly decreased BP probably by both reducing CO and causing vasodilatation, whereas etomidate only slightly decreased dBP and mBP without affecting other parameters. Propofol-induced changes were independent of LVEF or NYHA class. Induction of VF and internal defibrillation did not cause clinically significant changes apart from very modest drops in dBP and mBP values.

Key words: etomidate, propofol, internal electrical shock, haemodynamic parameters, impedance cardiography

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INTRODUCTION

Testing the efficacy of an implantable cardioverter-defibrillator (ICD) in terminating ventricular fibrillation (VF) is unpleasant and painful for a patient, and thus requires short sedation. Etomidate and propofol are the most widely used anaesthetic agents for this purpose [1]. Both drugs can alter haemodynamic parameters, with propofol being more prone to cause hypotension. However, the data on haemodynamic effects

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and mechanisms leading to hypotension are contradictory — some studies have shown profound differences between these two drugs [2–14], whereas others have not found any significant difference [15–22]. Moreover, data on mechanisms leading to drug-induced hypotension are scarce, especially in the setting of ICD testing. In particular, whether hypotension is due to reductions in cardiac contractility and cardiac output or due to vasodilatation has not yet been clearly established.

Medical understanding of haemodynamic effects of induced VF induction and subsequent internal defibrillation from ICD is also limited. Only a few studies have addressed this issue [23–25].

Impedance cardiography (ICG) is a non-invasive tool which can be used for the assessment of haemodynamic parameters in various settings [26–28]. Using this method, a whole range of haemodynamic parameters can be accurately measured. To date, ICG has been used for the assessment of propofol or etomidate-induced changes in haemodynamic parameters in only two small studies [29, 30], and only one study used this technique to examine the effects of internal electrical shock during ICD testing [31].

Accordingly, the aim of the present study was to address these issues.

METHODS

Study group

The study group consisted of 63 consecutive patients (mean age 66 ± 10 years, 51 males) who underwent ICD testing, either at the end of the implantation procedure or 2–3 days later before hospital discharge, and in whom an adequate quality of ICG recordings could be obtained. Demographic and clinical characteristics of the studied patients are presented in Table 1. This study was prospective, and the anaesthetic drugs were administered in a random order. The patients gave their informed written consent to participate in the study and the protocol of the study was accepted by the local Ethics Committee.

Table 1. Demographic and clinical characteristics of the studied patients

Parameter	Value
No. of patients	63
Age [years]	66 ± 10
Body mass [kg]	81 ± 16
Male/female	51/12
NYHA class 0–II	36 (57%)
NYHA class III–IV	27 (43%)
LVEF < 40%	49 (78%)
LVEF ≥ 40%	14 (22%)
Underlying cardiac disease:	
Coronary artery disease	49 (79%)
Dilated cardiomyopathy	7 (9%)
Hypertrophic cardiomyopathy	1 (2%)
Other	6 (10%)
Hypertension	35 (56%)
Aborted SCD	19 (30%)
Diabetes	20 (32%)
Chronic kidney disease	11 (17%)
Total etomidate dose [mg/kg]	0.15 ± 0.04
Total propofol dose [mg/kg]	1.07 ± 0.43

NYHA — New York Heart Association; LVEF — left ventricular ejection fraction; SCD — sudden cardiac death

Anaesthetic drugs

Patients were randomly assigned to etomidate (a slow bolus with a target dose of 0.3–0.4 mg/kg or propofol target dose of 2 mg/kg). Drug infusion was stopped when adequate anaesthesia was achieved, defined as unresponsiveness to commands and a loss of eyelid reflex. In a case of inadequate anaesthesia, repeated doses of drugs were administered.

ICD testing

After achieving adequate anaesthesia, VF was induced using 1 J shock on T wave or 50 Hz rapid ventricular pacing. The test shock was programmed to deliver energy 10 J lower than the maximal ICD output in order to ensure a defibrillation safety margin. In cases of ineffective shock and ongoing VF, maximal internal defibrillation shock was delivered. If this failed, external defibrillation was performed. In these cases, the position of the defibrillation lead was changed, reversed polarity was programmed, and ICD testing was repeated.

Impedance cardiography

The measurements were performed using commercially available equipment (Task Force Monitor Systems, CNSystems, Austria) which allows a non-invasive assessment of various haemodynamic parameters by measuring changes in the transthoracic electrical impedance. A detailed methodology has been described elsewhere [28]. In brief, the patients were studied while fasting after at least 30 min of rest. All the recordings were performed in the electrophysiology laboratory. In those patients in whom ICD testing was performed at the end of the implantation procedure, ICG electrodes were placed prior to the procedure. We used routine ICG electrodes configuration, placed at both sides of the thorax and at the neck base [28].

After a 15 min waiting period, needed for the stabilisation of haemodynamic status, the baseline ICG recording (ICG I) was obtained just before the initiation of anaesthetic drug injection. The next ICG recording (ICG II) was obtained after drug infusion, just before VF induction and defibrillation testing. The last ICG recording (ICG III) was obtained immediately after ICD testing which was usually possible 60–120 s after

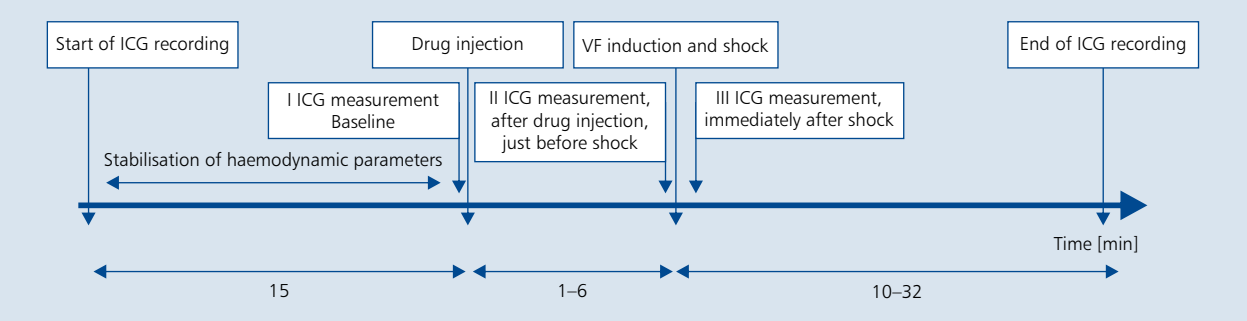

Figure 1. Design of the study; ICG — impedance cardiography; VF — ventricular fibrillation

electrical shock (the ICG system had to be switched off for the time of defibrillation). The design of the study is outlined in Figure 1. In summary, drug-induced changes in haemodynamic parameters were assessed comparing ICG I and ICG II values, and changes caused by VF and defibrillation — comparing the ICG II and ICG III results.

From each ICG recording period, 30 consecutive cardiac cycles were taken for the analysis, and the average of these measurements was used in the final analysis. Care was taken to include only good-quality signals and all the artefacts were excluded after a visual assessment of the recordings. The ICG data were presented in numerical values in the Excel programme. The following ICG parameters were measured [28]:

— Stroke volume (SV) [mL], calculated using the formula: $V_{EPT} \times (dZ_{max}/Z0) \times LVET$, where V_{EPT} is the part of the electrically participating thoracic volume, calculated from weight, height, age and gender, dZ_{max} is systolic amplitude [Ohm/s], Z0 stands for total thoracic impedance [Ohm] and LVET is the left ventricular ejection time [ms].
— Cardiac output (CO) [L/min], calculated using the formula: CO = SV × heart rate. Also, indexed CO called cardiac index (CI) [L/min/m^2] was calculated.
— Total peripheral resistance (TPR) [dyn×s/cm^5] calculated from the formula: (mBP – CVP)/CO × 80, where mBP is the mean arterial blood pressure [mm Hg] and CVP is central venous pressure. Also, indexed TPR (TPRI) was calculated [dyn×s×m^2/cm^5].

In addition, finger pletysmography was used to obtain beat-to-beat systolic (sBP) and diastolic (dBP) blood pressure values. Also mBP was calculated.

Statistical analysis

The results are presented as mean ± one standard deviation or numbers and percentages. Numerical values were compared using Student t-test or Mann-Whitney test, whereas qualitative parameters were compared using χ^2 test. The normality of data distribution was examined using Kolmogorov-Smirnov test. Drug-induced changes in haemodynamic parameters were compared using analysis of variance. A p value < 0.05 was considered significant.

RESULTS

All patients underwent ICD testing without any complication. The first ICD shock with preserved safety margin of 10 J was effective in 58 (92%) patients, in four patients maximal ICD shock, and in one — external defibrillation terminated VF. The ICG recordings were analysable in all 33 patients who received propofol, whereas in two patients from the etomidate group a high number of artefacts precluded meaningful analysis, leaving 30 patients in this subgroup available for further assessment.

A comparison of the demographic and clinical parameters between the propofol and etomidate groups is presented in Table 2. There were no significant differences between the groups.

A comparison of the haemodynamic parameters before and after anaesthetic injection is presented in Table 3. Propofol caused significant changes in all measured parameters. A decrease in BP was associated with reductions of SV and CO as well as a decrease in peripheral vascular resistance. Etomidate significantly decreased SV, whereas changes in the remaining parameters were small and non-significant.

A comparison of the drug-induced changes (Δ) in haemodynamic parameters is shown in Table 4. The relative decrease in the BP values was significantly greater following propofol than etomidate. Other parameters were also more affected following propofol infusion, although differences in Δ values failed to achieve statistical significance.

A comparison of the drug-induced changes in haemodynamic parameters according to New York Heart Association (NYHA) class and left ventricular ejection fraction (LVEF) is presented in Tables 5 and 6. There were no significant differences in the examined parameters between the groups.

The effects of VF induction and internal electrical shock on haemodynamic parameters are shown in Table 7. Apart from small but statistically significant decreases in dBP and mBP, other parameters did not change significantly.

DISCUSSION

The present study showed that: (1) propofol significantly decreased BP by both reducing CO and causing vasodilatation,

Table 2. Comparison of demographic and clinical parameters between patients allocated to propofol or to etomidate

Parameter	Propofol	Etomidate	P
No. of patients	33	30	NS (p = 0.705)
Age [years]	67 ± 11	66 ± 10	NS (p = 0.625)
Body mass [kg]	82.6 ± 17.7	78.5 ± 12.8	NS (p = 0.303)
Male/female	28/5	23/7	NS (p = 0.409)
NYHA class 0–II	19 (58%)	17 (57%)	NS (p = 0.942)
NYHA class III-IV	14 (42%)	13 (43%)	
LVEF < 40%	26 (79%)	23 (77%)	NS (p = 0.743)
LVEF ≥ 40%	7 (21%)	7 (23%)	
Underlying cardiac disease:			
Coronary artery disease	27 (82%)	22 (73%)	NS (p = 0.418)
Dilated cardiomyopathy	3 (9%)	4 (13%)	NS (p = 0.581)
Hypertrophic cardiomyopathy	1 (3%)	0 (0%)	NS (p = 0.962)
Other	2 (6%)	4 (13%)	NS (p=0.581)
Significant valvular defect	3 (9%)	1 (5%)	NS (p = 0.942)
Hypertension	19 (58%)	16 (53%)	NS (p = 0.735)
Aborted SCD	12 (36%)	7 (23%)	NS (p = 0.260)
Diabetes	8 (24%)	12 (40%)	NS (p = 0.180)
Chronic kidney disease	8 (24%)	3 (10%)	NS (p = 0.137)

Abbreviations as in Table 1

Table 3. Comparison of haemodynamic parameters before and after drug injection

	Before propofol (ICG I)	After propofol (ICG II)	P	Before etomidate (ICG I)	After etomidate (ICG II)	P
sBP [mm Hg]	123.36 ± 17.10	106.27 ± 18.02	0.000	118.49 ± 9.97	116.10 ± 18.33	NS (p = 0.453)
dBP [mm Hg]	83.70 ± 12.19	74.12 ± 13.76	0.000	82.00 ± 13.08	80.95 ± 17.54	NS (p = 0.667)
mBP [mm Hg]	93.90 ± 13.13	81.07 ± 16.11	0.000	91.72 ± 10.63	90.16 ± 17.19	NS (p = 0.564)
SV [mL]	61.10 ± 19.34	56.39 ± 15.67	0.003	60.59 ± 10.98	56.79 ± 10.13	0.022
CO [L/min]	4.51 ± 1.07	4.17 ± 0.73	0.003	4.30 ± 0.66	4.23 ± 0.74	NS (p = 0.548)
CI [L/min/m^2]	2.33 ± 0.60	2.16 ± 0.39	0.005	2.30 ± 0.42	2.27 ± 0.44	NS (p = 0.643)
TPR [dyn×s/cm^5]	1,735.76 ± 532.57	1,573.89 ± 390.52	0.020	1,722.90 ± 302.04	1,734.39 ± 405.58	NS (p = 0.876)
TPRI [dyn×s×m^2/cm^5]	3,374.99 ± 1,093.58	3,045.74 ± 818.20	0.013	3,253.54 ± 666.60	3,254.83 ± 770.68	NS (p = 0.993)

ICG — impedance cardiography; sBP — systolic blood pressure; dBP — diastolic blood pressure; mBP — mean blood pressure; SV — stroke volume; CO — cardiac output; CI — cardiac index; TPR — total peripheral resistance; TPRI — total peripheral resistance index

whereas etomidate only slightly decreased dBP and mBP without affecting other parameters; (2) propofol-induced changes were independent of LVEF or NYHA class; and (3) induction of VF and internal defibrillation did not cause clinically significant changes apart from a very modest drop in dBP and mBP values.

Haemodynamic effects of etomidate and propofol

These two anaesthetic agents have been compared in numerous studies. However, the literature does not favour either agent when benefits and side effects have been weighted. To date, in the setting of electrical shock, etomidate and propofol were compared only in patients undergoing electrical cardio-

Table 4. Comparison of drug-induced changes (Δ) in haemodynamic parameters

	Propofol (ICG I – ICG II)	Etomidate (ICG I – ICG II)	P
Δ sBP [mm Hg]	–17.09 ± 16.86	–2.39 ± 16.61	0.001
Δ dBP [mm Hg]	–9.58 ± 11.73	–1.06 ± 14.13	0.013
Δ mBP [mm Hg]	–12.84 ± 13.38	–1.56 ± 15.22	0.003
Δ SV [mL]	–4.71 ± 9.81	–3.80 ± 7.14	NS (p = 0.673)
Δ CO [L/min]	–0.34 ± 0.75	–0.07 ± 0.50	NS (p = 0.097)
Δ CI [L/min/m^2]	–0.17 ± 0.4	–0.03 ± 0.27	NS (p = 0.106)
Δ TPR [dyn×s/cm^5]	–161.87 ± 420.32	11.49 ± 344.43	NS (p = 0.087)
Δ TPRI [dyn×s×m^2/cm^5]	–329.25 ± 804.41	1.29 ± 644.67	NS (p = 0.086)

Abbreviations as in Table 3

Table 5. Comparison of changes in haemodynamic parameters following anaesthetic injection according to heart failure symptoms

	PROPOFOL			ETOMIDATE		
	NYHA 0–II (n = 19)	NYHA III–IV (n = 14)	P	NYHA 0–II (n = 17)	NYHA III–IV (n = 13)	P
Δ sBP [mm Hg]	–21.03 ± 14.11	–11.33 ± 19.36	NS (p = 0.291)	–7.23 ± 17.66	4.06 ± 13.11	NS (p = 0.078)
Δ dBP [mm Hg]	–12.32 ± 10.40	–5.57 ± 12.80	NS (p = 0.140)	–5.23 ± 12.99	4.51 ± 14.17	NS (p = 0.104)
Δ mBP [mm Hg]	–16.17 ± 11.60	–8.31 ± 14.71	NS (p = 0.308)	–6.28 ± 14.69	4.72 ± 14.09	NS (p = 0.063)
Δ SV [mL]	–4.71 ± 10.07	–4.72 ± 9.84	NS (p = 0.913)	–4.40 ± 6.02	–3.06 ± 8.52	NS (p = 0.511)
Δ CO [L/min]	–0.38 ± 0.83	–0.29 ± 0.65	NS (p = 0.716)	–0.16 ± 0.36	0.03 ± 0.63	NS (p = 0.483)
Δ CI [L/min/m^2]	–0.19 ± 0.46	–0.15 ± 0.33	NS (p = 0.743)	–0.07 ± 0.20	0.02 ± 0.34	NS (p = 0.599)
Δ TPR [dyn×s/cm^5]	–211.81 ± 436.44	–94.10 ± 403.17	NS (p = 0.382)	–65.81 ± 341.65	114.55 ± 334.28	NS (p = 0.486)
Δ TPRI [dyn×s×m^2/cm^5]	–417.30 ± 802.46	–209.75 ± 821.26	NS (p = 0.444)	–129.79 ± 666.77	176.07 ± 596.27	NS (p = 0.516)

Abbreviations as in Tables 1 and 3

version of atrial fibrillation (AF) [2–4, 8, 9, 15, 16], whereas no study addressed this issue in patients undergoing ICD testing. In the vast majority of AF studies, only drug-induced changes in BP were analysed and only in a few studies have other haemodynamic parameters been studied [5, 7, 17]. The majority of studies [2–14] have shown that propofol caused significant decreases in the mean and sBP and that this drop was significantly greater than following etomidate infusion. However, there are also reports showing that etomidate does not affect BP values at all, both in patients undergoing AF cardioversion [12, 32] and patients undergoing ICD implantation and testing [33]. In addition, there are also reports showing an increase in BP values following etomidate injection [4, 7]. Moreover, in eight reports, investigators failed to show any differences in etomidate or propofol-induced changes in BP values [15–22]. Even in these studies in which more detailed haemodynamic measurements were performed, results are not concordant. While Singh et al. [17] did not find any significant differences in the etomidate and propofol-induced changes in SV and CI, and Bendel et al. [5] — in CI and pulmonary capillary wedge pressure values, Ebert et al. [7] documented a significant fall in TPR following propofol injection.

In summary, the literature is not totally consistent, but it does usually show greater changes in BP following propofol rather than etomidate infusion. Our results are similar, although we investigated patients undergoing ICD testing and not patients undergoing electrical cardioversion of AF, which has not been reported so far.

Mechanisms leading to a propofol-induced fall in BP values have not been well established. Some studies have

Table 6. Comparison of changes in haemodynamic parameters following anaesthetic injection according to LVEF

	PROPOFOL			ETOMIDATE		
	LVEF < 40% (n = 26)	LVEF ≥ 40% (n = 7)	P	LVEF < 40% (n = 23)	LVEF ≥ 40% (n = 4)	P
Δ sBP [mm Hg]	–15.67 ± 16.27	–22.18 ± 19.26	NS (p = 0.480)	0.67 ± 12.29	–13.60 ± 25.75	NS (p = 0.179)
Δ dBP [mm Hg]	–8.27 ± 11.06	–14.26 ± 13.73	NS (p = 0.245)	1.74 ± 11.76	–11.32 ± 18.35	NS (p = 0.117)
Δ mBP [mm Hg]	–11.51 ± 12.61	–17.74 ± 16.04	NS (p = 0.628)	1.47 ± 12.11	–12.67 ± 21.12	NS (p = 0.131)
Δ SV [mL]	–5.48 ± 9.93	–1.89 ± 9.52	NS (p = 0.428)	–3.52 ± 7.00	–4.86 ± 8.24	NS (p = 0.706)
Δ CO [L/min]	–0.36 ± 0.76	–0.26 ± 0.76	NS (p = 0.860)	–0.03 ± 0.52	–0.22 ± 0.41	NS (p = 0.333)
Δ CI [L/min/m^2]	–0.18 ± 0.40	–0.15 ± 0.43	NS (p = 0.965)	–0.01 ± 0.28	–0.12 ± 0.22	NS (p = 0.236)
Δ TPR [dyn×s/cm^5]	–116.46 ± 433.46	–330.52 ± 342.45	NS (p = 0.234)	52.67 ± 288.49	–139.51 ± 506.20	NS (p = 0.695)
Δ TPRI [dyn×s×m^2/cm^5]	–248.86 ± 836.39	–627.86 ± 635.76	NS (p = 0.333)	78.16 ± 511.51	–280.55 ± 1,011.93	NS (p = 0.737)

Abbreviations as in Tables 1 and 3

Table 7. Comparison of haemodynamic parameters before (ICG II) and immediately after (ICG III) electrical shock

	ICG II	ICG III	P
sBP [mm Hg]	110.31 ± 19.24	107.51 ± 23.66	NS (p = 0.141)
dBP [mm Hg]	76.95 ± 16.07	72.88 ± 17.52	0.002
mBP [mm Hg]	85.23 ± 17.24	81.76 ± 19.71	0.017
SV [mL]	56.58 ± 13.26	58.62 ± 17.31	NS (p = 0.215)
CO [L/min]	4.20 ± 0.73	4.21 ± 0.97	NS (p = 0.940)
CI [L/min/m^2]	2.21 ± 0.41	2.20 ± 0.47	NS (p = 0.886)
TPR [dyn×s/cm^5]	1,641.37 ± 402.77	1,573.55 ± 443.98	NS (p = 0.276)
TPRI [dyn×s×m^2/cm^5]	3,124.93 ± 792.87	2,998.47 ± 834.47	NS (p = 0.276)

Abbreviations as in Table 3

suggested that negative inotropic effect resulting in decreased CO is the main mechanism responsible for hypotension [34], whereas in other reports vasodilatation and reduced TPR were found to be more important [35, 36]. Our study suggests that in patients with ICD who are usually more ill than patients with AF undergoing cardioversion, both mechanisms are involved.

The postulated mechanisms leading to propofol-induced vasodilatation are numerous and involve endothelial activation of nitric oxide production [35], direct effects of propofol on smooth muscles in vascular wall, regulated by KATP channels [35] as well as vascular relaxation independent of endothelium [36]. Other mechanisms responsible for propofol-induced hypotension include the attenuation of adrenergic drive and the impairment of baroreceptor reflex [7].

Etomidate vs. propofol and cardiac dysfunction

The results of our study suggest that drug-induced changes are similar in patients with preserved and reduced LVEF as well as in patients with various degrees of heart failure. However, bearing in mind that propofol induces hypotension more frequently than etomidate, the latter agent is usually preferred in patients with baseline low BP or with haemodynamic instability. Propofol, however, has the advantage over etomidate in terms of a faster return to baseline status following anaesthesia. This is important in some clinical situations such as patients with chronic obstructive pulmonary disease. Camci et al. [37] also reported propofol-induced significant hypotension occurring in patients undergoing ICD testing, although in their study a clinically significant BP decrease was

noted only in patients with LVEF < 30%. This may suggest that in patients with markedly reduced systolic LV function, etomidate should be the preferred anaesthetic agent even in patients with normal baseline BP values. However, in our study we did not observe any clinically significant hypotension, even in patients with LVEF ≤ 30% — there were 26 such patients. Thus, we believe that in stable patients on optimal pharmacological therapy, propofol is safe during ICD testing even in patients with low LVEF, and can be used either by anaesthesiologists or cardiologists because of the ease of drug administration, prompt resolution of anaesthesia upon drug infusion termination, and lack of side effects typical of etomidate such as tremor and seizures.

Effects of VF induction and electrical shock on haemodynamic variables

ICD testing is generally regarded as a safe procedure. However, it can lead in some patients to delayed normalisation of BP and CO due to myocardial stunning and decreased sympathetic activity [24, 38, 39], especially in those with very low LVEF. Reports in the literature concerning haemodynamic changes during ICD testing are surprisingly scarce. Meyer et al. [23], using invasive measurements in the pulmonary artery in 11 patients with low LVEF, failed to document any significant drop in CO in patients following ICD testing. Contradictory results were presented by Skhirtladze et al. [24], who found that invasively measured CO and mBP decreased in some patients, especially in those with LVEF < 30%. Also Toh et al. [25] showed that patients with LVEF < 45% had a transient decrease in systolic left ventricular function due to electrical shock, whereas patients with LVEF > 45% did not. The results of these two studies suggest that the lower the baseline LVEF, the more negative are the effects of shock on haemodynamic stability. Our results do not support these findings and are in line with Krzesiński et al. [31] who also used ICG for the assessment of ICD shock-induced changes in haemodynamic parameters, and did not find any clinically significant complications in their cohort of patients with a mean LVEF of 30.7 ± 9.5%.

Our findings obviously do not prove that ICD testing is not associated with any risk. There are case reports on such complications related to ICD testing as cardiogenic shock, pulmonary oedema or death [40, 41]. Also, inadvertent termination of AF following ICD shock can lead to stroke in patients who are not adequately anticoagulated. However, it seems that in stable and optimally treated patients, ICD testing is safe and does not cause significant alterations in haemodynamic status. Although in recent years there has been a trend towards abandoning ICD testing because the risk of high defibrillation threshold is low (2–3%) [42], the majority of investigators believe that this procedure should be performed, especially in those with a high risk of increased defibrillation threshold or high probability of arrhythmia recurrences [43, 44].

Difficulties in comparing the results of our study to published data

First of all, we used non-invasive ICG to investigate haemodynamic parameters, which had previously only been done in one study for ICD testing [31] and only in two small studies examining separately the effects of etomidate or propofol during anaesthesia [29, 30]. Although ICG is a reproducible and well-validated technique [45], some differences between ICG and invasive measurements may exist. Secondly, there are numerous differences between published studies as far as initial anaesthetic drug doses, rate of infusion, final doses, and time points of drug effects evaluation are concerned. In addition, in many reports other drugs such as fentanyl were also used for the induction of anaesthesia, which may influence haemodynamic parameters. The total dose of etomidate and propofol in our study was slightly lower than in some other reports, which also might have influenced the results.

Limitations of the study

Firstly, there are limitations of ICG itself. Although the method has been validated, it is based on indirect measurements and some parameters such as TPR and CO are calculated based on the values of other indices like SV, BP and heart rate. In our study, we did not use any other invasive or non-invasive method for evaluation of haemodynamic status. Thus, conclusions regarding the mechanisms of propofol-induced hypotension are limited.

Secondly, the quality of recordings is not always perfect, and in our study two patients were not included in the analysis due to the high number of artefacts. In addition, a 30 cycles period was arbitrarily chosen for analysis, and may not be optimal.

Thirdly, we assumed that no other factors affected haemodynamic parameters when assessing the effects of ICD shock on haemodynamics. We believe that this holds true because the time period between ICG II and ICG III measurement was relatively short (in the range of 1–2 min); however, small changes in the anaesthetic drugs blood concentration or other factors affecting the haemodynamic system cannot be excluded.

Fourthly, although the number of studied patients was sufficient to perform a statistical comparison of the effects of drugs and ICD shock on examined parameters, it was too low to conduct meaningful subgroup analysis, and thus conclusions regarding patients with preserved LVEF are limited.

Finally, the effects of ICD shock were not examined immediately after the shock but 30–50 s later, after turning on the ICG machine which had to be shut down during ICD discharge. Thus, we may have omitted the very early changes in haemodynamic parameters caused by ICD shock. There is no doubt that VF causes profound haemodynamic changes, but they were short lasting in our patients and ICG measurements obtained > 30 s from this event returned to pre-test values.

CONCLUSIONS

Propofol significantly decreased BP probably by both reducing CO and causing vasodilatation, whereas etomidate only slightly decreased dBP and mBP without affecting other parameters. Propofol-induced changes were independent of LVEF or NYHA class. Induction of VF and internal defibrillation did not cause clinically significant changes apart from very modest drops in dBP and mBP values.

Conflict of interest: *none declared*

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Efekty hemodynamiczne etomidatu, propofolu i elektrowstrząsu u chorych poddawanych testowaniu kardiowertera-defibrylatora

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Streszczenie

Wstęp: Leki anestetyczne i indukcja migotania komór (VF) oraz następowy elektrowstrząs mogą ujemnie wpływać na stan hemodynamiczny chorych, u których testuje się skuteczność kardiowertera-defibrylatora (ICD). Mechanizmy oddziaływania na parametry hemodynamiczne (ujemny efekt inotropowy *vs*. wazodylatacja) etomidatu i propofolu nie są do końca zbadane, podobnie jak skutki hemodynamiczne indukcji VF i następowego wyładowania z ICD. Kardiografia impedancyjna nie była jeszcze używana do oceny skutków działania leków anestetycznych.

Cel: Celem pracy było porównanie efektów hemodynamicznych etomidatu i propofolu oraz wywołania VF i następowego elektrowstrząsu u chorych z ICD.

Metody: Do prospektywnego badania z randomizacją włączono 63 kolejnych chorych (średni wiek 66 ±10 lat, 51 mężczyzn) poddawanych testowaniu skuteczności ICD. Parametry hemodynamiczne mierzono przy użyciu kardiografii impedancyjnej (Task Force Monitor Systems, CNSystems, Austria) przed i po podaniu etomidatu (n = 30) lub propofolu (n = 33) oraz natychmiast po elektrowstrząsie przerywającym wyindukowane VF. Rejestrowano ciśnienie skurczowe (sBP), rozkurczowe (dBP) i średnie (mBP), pojemność wyrzutową (SV), rzut serca (CO) i całkowity opór obwodowy (TPR).

Wyniki: Propofol istotnie obniżył wszystkie mierzone parametry (sBP: 123,4 ± 17,1 *vs*. 106,3 ± 18 mm Hg; $p < 0,0001$; dBP: 83,7 ± 12,2 *vs*. 74,1 ± 13,8 mm Hg; $p < 0,0001$; mBP: 93,9 ± 13,1 *vs*. 81,1 ± 16,1 mm Hg; $p < 0,0001$; SV: 61,1 ± 19,3 *vs*. 56,4 ± 15,7 ml; $p < 0,003$; CO: 4,51 ± 1,07 *vs*. 4,17 ± 0,73 l/min; $p < 0,003$; TPR: 1735,8 ± 532,6 *vs*. 1573,9 ± 390,5 dyn × s/cm^5), podczas gdy etomidat obniżył istotnie jedynie SV (60,6 ± 11 *vs*. 56,8 ± 10 ml; $p < 0,022$). Zmiany hemodynamiczne po propofolu były podobne u chorych z obniżoną (< 40%) i zachowaną (≥ 40%) frakcją wyrzutową lewej komory oraz u osób z różnym stopniem niewydolności serca (NYHA I–II *vs*. III–IV) (NS). Wywołanie VF i elektrowstrząs z ICD nie spowodowały wyraźnych zmian hemodynamicznych poza istotnym statystycznie, ale niewielkim spadkiem dBP i mBP (odpowiednio, 77 ± 2 *vs*. 72,9 ± 18 mm Hg; $p < 0,002$ i 85,2 ± 17 *vs*. 81,8 ± 20 mm Hg; $p < 0,017$). Podczas testowania ICD nie stwierdzono żadnych powikłań.

Wnioski: Propofol istotnie obniżył ciśnienie tętnicze prawdopodobnie zarówno poprzez zmniejszenie SV, jak i wazodylatację (obniżenie TPR), niezależnie od stopnia niewydolności serca lub uszkodzenia lewej komory. Po etomidacie pogorszenie parametrów hemodynamicznych było znacznie mniejsze, co potwierdza tendencję do preferowania tego leku w stosunku do propofolu u chorych niestabilnych hemodynamicznie lub z niskim ciśnieniem. Indukcja VF i elektrowstrząs nie wpłynęły istotnie na hemodynamikę układu sercowo-naczyniowego.

Słowa kluczowe: propofol, etomidat, elektrowstrząs, kardiografia impedancyjna

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