Cardiac Arrest During Spine Surgery in the Prone Position: Case Report and Review of the Literature

Victor E. Staartjes[1,2], Shiva A. Schillevoort[3], Patricia G. Blum[2], J. Peter van Tintelen[4], Wouter E. Kok[3], Marc L. Schröder[1]

Key words
- Cardiac arrest
- Catecholaminergic polymorphic ventricular tachycardia
- Genetic
- Lumbar fusion
- Risk factor
- Spine
- Ventricular fibrillation

Abbreviations and Acronyms
ASA: American Society of Anesthesiologists
CA: Cardiac arrest
CPR: Cardiopulmonary resuscitation
CPVT: Catecholaminergic polymorphic ventricular fibrillation
CT: Computed tomography
ECG: Electrocardiogram
EST: Epinephrine stress test
ROSC: Return of spontaneous circulation
VAS: Visual analogue scale
VF: Ventricular fibrillation

From the [1]Department of Neurosurgery, Bergman Clinics, Amsterdam, The Netherlands; [2]Faculty of Medicine, University of Zurich, Zurich, Switzerland; [3]Department of Cardiology, Academic Medical Center, Amsterdam, The Netherlands; and [4]Department of Clinical Genetics, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands

To whom correspondence should be addressed: Victor E. Staartjes, B.Med. [E-mail: victor.staartjes@gmail.com]

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OBJECTIVE: **Intraoperative cardiac arrest (CA) is usually attributable to pre-existing disease or intraoperative complications. In rare cases, intraoperative stress can demask certain genetic diseases, such as catecholaminergic polymorphic ventricular tachycardia (CPVT). It is essential that neurosurgeons be aware of the etiologies, risk factors, and initial management of CA during surgery with the patient in the prone position.**

METHODS: **We present a case of CA directly after spinal fusion for lumbar spondylolisthesis and review the literature on cardiac arrests during spinal neurosurgery in the prone position. We focus on etiologies of CA in patients with structurally normal hearts.**

RESULTS: **After resuscitation, a 53-years-old female patient achieved return of spontaneous circulation after 17 minutes, without any neurologic deficits and with substantial improvement of functional disability and pain scores. Extensive imaging, stress testing, and genetic screening ruled out common etiologies of CA. In this patient with a structurally normal heart, CPVT was established as the most likely cause. We identified 18 additional cases of CA associated with spinal neurosurgery in the prone position. Most cases occurred during deformity or fusion procedures. Commonly reported etiologies of CA were air embolism, hypovolemia, and dural traction leading to vasovagal response. In patients with structurally normal hearts, inherited arrhythmia syndromes including CPVT, Brugada syndrome, and long QT syndrome should be included in the differential diagnosis and specifically included in testing.**

CONCLUSIONS: **Although intraoperative CA is rare during spine surgery, neurosurgeons should be aware of the etiologies and the specific difficulties in the management associated with the prone position.**

INTRODUCTION

Cardiac arrest (CA) is an exceedingly rare complication in noncardiac surgery. The incidence is approximately 0.7% in cardiac surgery, and there are only a few reported cases of CA associated with spinal neurosurgery.[1] Overall, the incidence of perioperative cardiac events is 6.7 per 1000 lumbar spine procedures, but only a minority of those are cardiac arrests.[2] The outcome of in-hospital CA is estimated to be lethal in 77% of cases, and there is a high risk for new neurological morbidity.[3] In recent years, the incidence of intraoperative and perioperative CA associated with noncardiac surgery has decreased markedly with the advent of improved technologies and changing clinical practices.[4] Nonetheless, neurosurgeons should be aware of the incidence, risk factors, and of the most effective management strategies of this grave complication. Especially in spinal surgery with the patient in prone position, management of CA is linked to additional difficulties owing to positioning, hemodynamics, and sometimes posterior instrumentation. We report a case of CA directly after a lumbar fusion procedure. In addition, we review the literature for other cases of CA associated with spinal neurosurgery, and we summarize the evidence regarding risk factors and effective management strategies.

CASE PRESENTATION

Preoperative Course

A 53-year-old female patient came to our outpatient spine clinic with chronic lower back and leg pain, both with a Visual Analogue Scale (VAS) severity of 70. Magnetic resonance imaging showed a grade 1 spondylolisthesis at L5-S1. The patient was athletic with a body mass index of 22.1 kg/m^2 and an American Society of Anesthesiologists (ASA) score of 2

Table 1. Blood Tests Before, During, and After the Complication

Measurement	Reference Range	1 month Preoperatively	During Complication	2 hours after Complication	2 weeks After Complication
Sodium (mmol/L)	135–145	142	141	141	141
Potassium (mmol/L)	3.5–5.0	4.9	3.9	3.9	4.0
Chloride (mmol/L)	96–106	—	109*	106	—
Urea (mmol/L)	2.1–7.1	3.1	5.3	4.7	3.3
Glucose (mmol/L)	3.5–11.1	4.4	14.4*	15.1*	—
eGFR (mL/mol/L)	>60	>90	—	64	>90
Creatinine (μmol/L)	53–97	61	—	89	49*
Hematocrit (L/L)	0.36–0.48	—	0.36	0.34*	—
Hemoglobin (mmol/L)	7.3–9.8	7.8	7.6	6.4*	5.6*
Leukocytes (10^9 cells/L)	3.5–11.0	8.4	—	24.2*	—
Thrombocytes (10^3 cells/L)	150–450	—	—	285	—
ALT (U/L)	7–56	—	—	222*	—
CRP (mg/L)	0–10	1	—	1.5	20.1*
Troponin T (ng/mL)	<0.01	—	—	0.008	—

eGFR, estimated glomerular filtration rate; ALT, alanine aminotransferase; CRP, C-reactive protein.
*Values outside the reference range.

because of chronic bronchitis and essential hypertension, for which she received candesartan (8 mg). She was evaluated for palpitations at the age of 39 years, and at the time demonstrated premature ventricular extrasystoles during an exercise test and at Holter monitoring, both of which were no longer available. She did not receive medication at the time. In 2007, the patient underwent reduction mammaplasty uneventfully. The preoperative electrocardiogram (ECG) was inconspicuous at a resting sinus heart rate of 64 beats/minute. Blood testing revealed no abnormalities (Table 1). The patient gave written informed consent for this report.

Surgical Course

Preoperatively, the patient received cefazolin (2000 mg) as antibiotic prophylaxis, and general anesthesia was maintained using propofol and sufentanil. The patient underwent robot-guided minimally invasive transforaminal interbody fusion at L5-S1. The procedure was performed as described before with the patient in prone position and with a barrel-shaped cushion positioned under the pelvis to augment lordosis.[5] Intraoperatively, the right S1 pedicle screw had to be repositioned, and a bicortical trajectory was chosen for optimal bony purchase (Figure 1). The duration of the procedure was 144 minutes, and the patient's condition remained stable with unsuspicious monitoring throughout the entire procedure. Intraoperatively, the patient received ephedrine in 6 boluses of 2.5 mg. Estimated blood loss was 200 mL.

After wound closure, the patient's legs appeared cyanotic and had a marmorated pattern. This is not an uncommon sight owing to the pelvic cushion sometimes restricting venous return. ECG leads were briefly disconnected for transfer of the patient from the operating table onto the bed, during which the patient was rotated from prone to supine positioning. Leg cyanosis disappeared immediately, indicating restoration of perfusion. The patient's pulse steadily increased from 60 to more than 100 beats/minute during surgery.

Onset of the Complication

The patient appeared greyish after rotation, and atropine (0.5 mg) and ephedrine (10 mg) were administered to mitigate the possibility of hypotension. Monitoring leads were reattached immediately, and the ECG appeared asystolic. No radial pulse was present.

Course of Treatment

Manual cardiopulmonary resuscitation (CPR) was started immediately, and an external defibrillator was attached. We followed the guidelines of the European Resuscitation Council.[6] The patient remained intubated and underwent mechanical ventilation. The ECG showed a regular broad QRS rhythm of 90 beats/minute (Figure 2A), which changed into a bradycardic sinus rhythm of 40 beats/minute (Figure 2B). Upon injection of ephedrine, the rhythm converted to ventricular fibrillation (VF; Figure 2C). The first shock (200 J) was delivered, followed by sinus bradycardia with somewhat smaller QRS complexes (Figure 2D), but with low CO_2 on the capnogram. One milligram of epinephrine was given twice. Two minutes after the first VF episode, ventricular fibrillation recurred (Figure 2E). Amiodarone (150 mg) was administered, and the second defibrillation shock was delivered (200 J). Sinus rhythm was restored with extrasystole (Figure 2F). Rising CO_2 was observed on capnography, and the return of a radial pulse confirmed the return of spontaneous circulation (ROSC). Overall, the patient was resuscitated over the course of 17 minutes, with 2

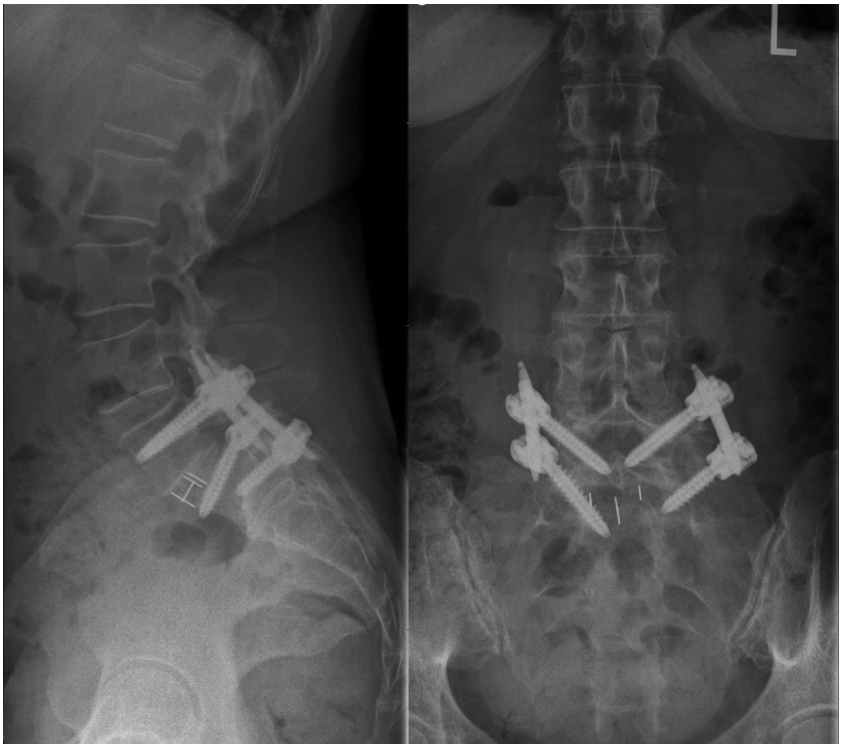

Figure 1. Postoperative radiographs demonstrating successful reduction of spondylolisthesis at L5-S1. Note the revised right S1 pedicle screw with a bicortical trajectory.

defibrillations being administered close together.

During resuscitation, a blood sample was taken from the femoral vein (Table 1). Apart from high glucose and slightly elevated chloride, the sample appeared inconspicuous. The patient was transferred to an academic medical center with adequate intensive care facilities. There, the hypothermic patient was kept under general anesthesia until an adequate body temperature was reached. Computed tomographic scan images and echography of the heart, abdomen, and thorax were performed, and coronary artery disease, retroperitoneal hematoma, and pulmonary embolisms were ruled out. The cardiac ventricular walls appeared normal apart from slight left ventricular hypertrophy on echocardiography, and the coronary arteries appeared normal on a computed tomographic angiogram. No free fluid was spotted during abdominal echography. A blood sample was taken 2 hours after onset of the complication (Table 1). Laboratory tests were repeated, demonstrating no signs of cardiac damage. A declining hemoglobin level was observed, but without signs of bleeding on repeated imaging of the abdomen and retroperitoneum. After clinical stabilization, contrast-enhanced magnetic resonance imaging of the heart revealed no abnormalities. Two weeks after presentation, an epinephrine stress test (EST) was completed. During the EST, the patient developed sinus tachycardia with ventricular extrasystoles with 2 distinct morphologies (both with a right bundle branch block morphology), and a single doublet of ventricular extrasystoles with first a right bundle branch block morphology and the second a changed morphology arising from the T wave of the first ventricular extrasystole. The EST was counted as abnormal, indicating a high likelihood for catecholaminergic polymorphic ventricular tachycardia (CPVT). The patient was prescribed metoprolol (100 mg daily). The patient developed no abnormal rhythms during exercise testing with metoprolol.

Genetic Screening

A targeted next-generation sequencing panel was used to identify any mutations associated with channelopathies and some cardiomyopathies associated with CA and sudden cardiac death. Genes underlying CPVT, Brugada syndrome, long QT syndrome, short QT syndrome, and other channelopathies were screened. Specifically, *ABCC9*, *AKAP9*, *ANK2*, *ASPH*, *CACNA1C*, *CACNA1D*, *CACNA2D1*, *CACNB2*, *CALM1*, *CALM2*, *CALM3*, *CASQ2*, *CAV3*, *DPP6*, *GJA5*, *GPD1L*, *HCN4*, *JPH2*, *KCNA5*, *KCND3*, *KCNE1*, *KCNE5*, *KCNE2*, *KCNE3*, *KCNH2*, *KCNJ2*, *KCNJ5*, *KCNJ8*, *KCNQ1*, *LAMP2*, *LMNA*, *NKX2-5*, *NPPA*, *PKP2*, *PLN*, *PPA2*, *PRKAG2*, *RANGRF*, *RYR2*, *SCN1B* *SCN2B*, *SCN3B*, *SCN4B*, *SCN5A*, *SCN10A*, *SLMAP*, *SNTA1*, *TNNT2*, *TRDN*, and *TRPM4* were screened. No abnormalities were identified.

Outcome

At discharge from the hospital, the patient showed no neurologic deficits. At 6-week follow-up, the patient indicated a 5-dimension EuroQOL (EQ-5D) index of 0.552, an EQ-5D VAS score of 70, VAS back pain score of 20, VAS leg pain score of 0, Oswestry Disability Index of 12, and Roland-Morris Disability Questionnaire score of 5.

DISCUSSION

Case Discussion

Multiple causes come into question in this case of CA directly after a lumbar fusion procedure. The first rhythm obtained is asystole, and shortly after that the ECG pads are applied again with the appearance of a broad-based regular tachycardia, possibly ventricular tachycardia. It is presumed that epinephrine might have caused the exacerbated rhythm disturbance, leading to ventricular fibrillation; however, ventricular fibrillation is not expected to occur with normal doses of epinephrine, except in patients with increased sensitivity for epinephrine, such as patients with CPVT. To date, no explicit etiology has been identified. The absence of structural heart disease suggests an "electrical" heart disease. The positive EST with ventricular extrasystoles during epinephrine infusion, which in addition were blocked during an exercise

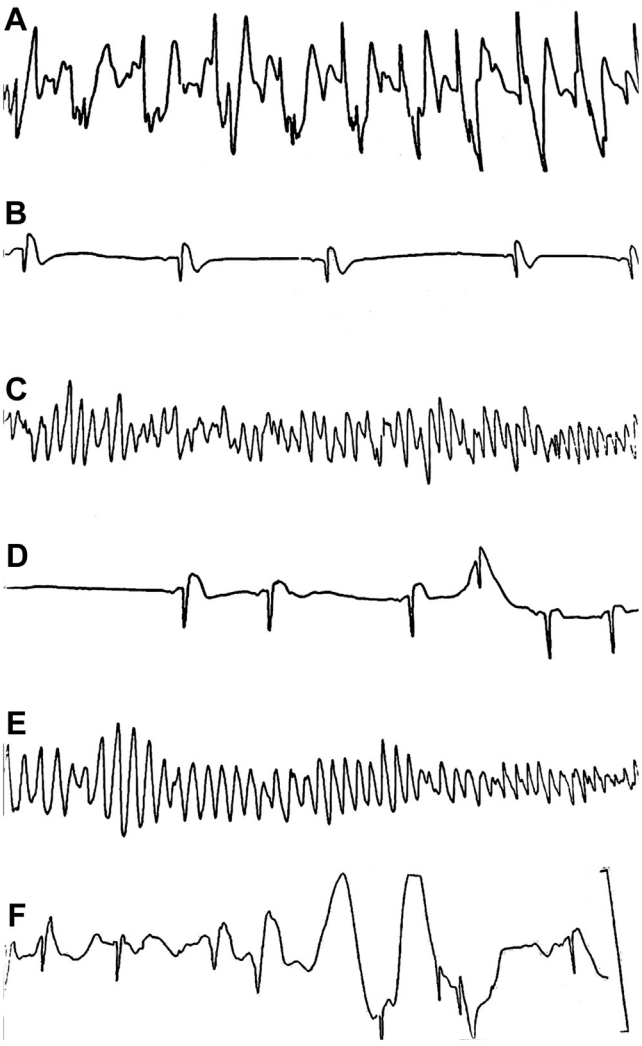

Figure 2. Electrocardiographic records from the defibrillator. (**A**) Regular broad QRS rhythm (90 beats/minute) without discernable P waves and interspersed probably resuscitation artefacts. (**B**) Broad-based QRS rhythm in (**A**) converts into sinus bradycardia of 40 beats/minute, with normal AV conduction and QRS width smaller than that in (**A**). (**C**) Ventricular fibrillation. Onset was not detected and was followed by a 200-J defibrillation shock. (**D**) Irregular sinus beats with normal AV conduction and QRS width slightly smaller compared to those in (**B**). (**E**) Ventricular fibrillation. Onset was not detected and was followed by a 200-J defibrillation shock. (**F**) Sinus rhythm with similar width and shape of QRS complexes shown in (**D**).

test under metoprolol, suggests a high probability of CPVT. The EST exhibits high specificity yet low sensitivity.[7] The assumption of CPVT is further corroborated by previous episodes of palpitations during periods of stress and exercise and a single doublet of ventricular extrasystole with two morphologies. No genetic abnormalities were detected; however, no causative mutation is identified in many patients with CPVT.[8]

As a differential diagnosis to explain the first episode of asystole, deep venous thrombosis and subsequent pulmonary or coronary embolisms could be suspected because of the usual compression of the iliac veins by the barrel-shaped cushions used for patient positioning. The patient's legs showed cyanosis before the onset of the complication, which disappeared after the patient was rotated; however, postoperative imaging ruled out thromboembolic causes.[9,10] During intraoperative revision of the right S1 pedicle screw, we opted for a bicortical trajectory. This renders possible a hypovolemic etiology of the VF and subsequent loss of hemoglobin, caused by vascular laceration and retroperitoneal hematoma. Multiple imaging modalities were repeatedly used, but no free fluid was detected.

The patient was successfully resuscitated in the supine position. ROSC was confirmed after 17 minutes and 2 administered shocks. Neurologic outcome was excellent. CPVT appears to be the most likely etiology.

Review of the Literature

After a thorough review of the literature, 18 other cases of CA associated with spine surgery in the prone position were identified (**Table 2**).[9-15,17-23] The incidence of intraoperative and perioperative CAs in spine surgery ranges between 4.3 and 21.3 per 10,000 cases.[4,24,25] Other cardiac complications occur in approximately 0.7% of lumbar spinal procedures.[2] Regional anesthesia lowers the rate of cardiac complications; however, this is most likely attributable to most high-risk procedures being performed using general anesthesia.[24] Intraoperative CAs dramatically reduce immediate and in-hospital survival to less than 37%.[2,4,24,25] Overall, independent predictors of death after intraoperative CA include hemorrhage, hypotension before the arrest, nonstandard working hours, need for vasopressor or inotropic support, and duration of the CA greater than 15 minutes.[24,26]

General risk factors for intraoperative and perioperative CAs associated with spine surgery have been identified in the literature. In an analysis of the American College of Surgeons National Surgical Quality Improvement Program database, ASA class 4 or 5 and black African or Asian ethnicity showed a higher incidence of CA.[25] An analysis of half a million lumbar spine procedures by Fineberg et al.[2] identified lumbar fusion, age over 65 years, acute and chronic blood loss anemia, deficiency anemia, male sex, obesity, coagulopathy, and a range of structural heart diseases as independent risk factors for cardiac complications during spine surgery. Blood transfusion and preoperative independence have also

Table 2. Cases of Cardiac Arrests Associated With Spinal Neurosurgery in the Prone Position From the Literature

References	Age	Sex	Indication	Intervention	Level	Rhythm	Etiology	CPR Position	ROSC
Albin et al., 1991[11]	40	Male	Lumbar disc herniation	Fusion	L3-L4	Asystole	Air embolism	Supine	No
Albin et al., 1991[11]	40	Male	Lumbar disc herniation	Fusion	L5-S1	Asystole	Air embolism	Supine	No
Chauhan et al., 2016[10]	49	Male	Lumbar disc herniation	Discectomy	L4-L5	Asystole	Vasovagal reaction owing to dural traction	Prone	Yes
Chen et al., 2002[12]	75	Female	Compression fracture	Percutaneous vertebroplasty	L2 and L4	Asystole	Pulmonary embolism	Supine	No
Deschamps et al., 2004[13]	37	Female	Stenosis	Laminectomy	L5-S1	Severe bradycardia	Vasovagal reaction due to electric stimulation of parasympathetic nerve endings with electrocautery	ROSC after atropine and ephedrine	Yes
Ewah et al., 1991[14]	26	Female	Lumbar disc herniation	Discectomy	L4-L5	—	Aortic laceration	Supine	No
Hong et al., 2017[15]	62	Female	Spondylolisthesis	Fusion	L4-S1	Asystole	Pulmonary embolism	Supine	Yes
Lang et al., 1989[16]	16	Male	Duchenne muscular dystrophy	Deformity	—	—	Air embolism	Supine	No
López et al., 1999[17]	15	Female	Progressive scoliosis	Fusion	—	Asystole	Wound irrigation with hydrogen peroxide leading to gas embolism	—	Yes
Mallick et al., 2013[18]	18	Female	Wedge fracture	Fusion	L2-L4	Ventricular tachycardia	Anaphylactic Shock (Atracurium)	Prone	Yes
Mandal, 2004[9]	36	Male	Lumbar disc herniation	Discectomy	L4-L5	Severe bradycardia	Vasovagal reaction owing to dural traction	ROSC after atropine	Yes
McCarthy et al., 1990[19]	18	Female	Idiopathic scoliosis	Deformity	—	—	Air embolism	—	No
McCarthy et al., 1990[19]	15	Male	Congenital scoliosis	Deformity	—	—	Air embolism	—	No
McDouall et al., 2007[20]	43	Female	Lumbar spinal stenosis	Laminectomy	L4-L5	Cardiac Arrest	Air embolism	Supine	No
Reid et al., 1999[21]	15	Male	Duchenne muscular dystrophy	Deformity	—	Ventricular fibrillation	Cardiomyopathy	Prone	Yes
Sutherland et al., 1997[22]	8	Female	Progressive scoliosis	Deformity	T1-S1	Asystole	Air embolism	Prone	No
Sutherland et al., 1997[22]	12	Female	Progressive scoliosis	Deformity	T2-S1	Asystole	Air embolism	Supine	No
Tobias et al., 1994[23]	12	Male	Progressive scoliosis	Deformity	—	Asystole	Hypovolemic shock	Prone	Yes

CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation.

demonstrated a risk for intraoperative CA.[2,4]

Surgical Risk Factors

Most cases in the literature occurred during deformity or spinal fusion procedures. Although gender was approximately evenly distributed, CAs occurred at a relatively young mean age of 29.8 ± 18.5 years, which is partly explained by the high proportion of adolescent deformity correction procedures. Of the 18 cases, 13 patients (72%) underwent CPR, of which 5 patients (38%) remained in the prone position. Eight patients (44%) showed ROSC and survived (Table 2).

The differential diagnosis of cardiovascular instability during surgery includes hypovolemic causes, which can be of absolute (underestimated blood and fluid loss, concealed blood loss [e.g., retroperitoneal, free peritoneal, pericardial, pleural]) or relative nature (spinal shock secondary to cord trauma or traction, anaphylaxis or anaphylactoid reaction, or transfusion reaction).[16] Other causes can include arrhythmia owing to channelopathy, cardiomyopathy, or mechanical vessel obstruction (improper positioning, pericardial tamponade,

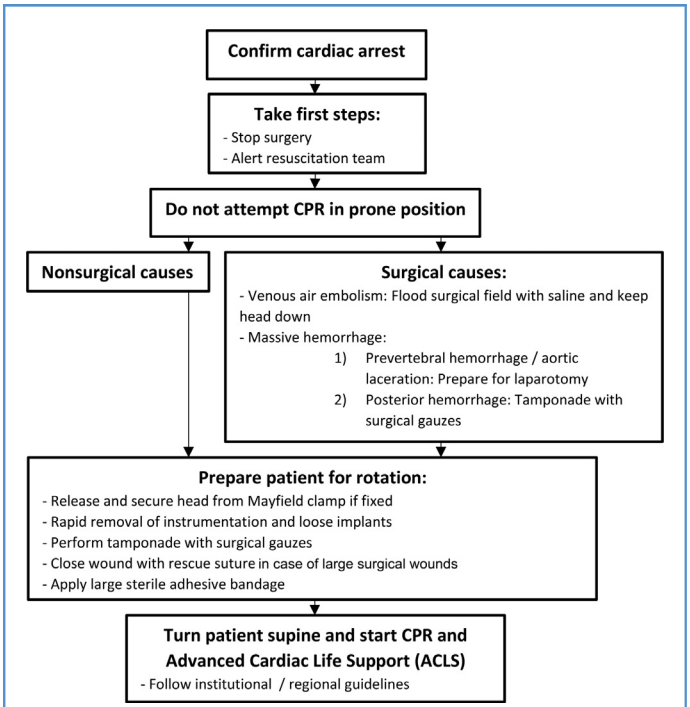

Figure 3. Flowchart demonstrating a recommendation for the steps that neurosurgeons should take if cardiac arrest is suspected during spine surgery in the prone position. (Modified and adapted from the guidelines of the U.K. Resuscitation Council and Society of British Neurological Surgeons.[34]) For cardiopulmonary resuscitation (CPR) and advanced cardiac life support, institutional guidelines or those of the American Heart Association[33] or European Resuscitation Council[6] should be followed.

tension pneumothorax, air or fat embolism).[16]

Eight (44%) of 18 cases we identified from the literature occurred because of gas embolisms. Venous air embolisms can lead to subclinical emboli or fatal massive emboli.[20] A gravitational gradient between the site of surgery and the right ventricle is a prerequisite for a venous air embolism. It has been shown that air embolisms occur after a change in patient positioning.[20] Although venous air embolisms appear to be unlikely during spinal surgery because of a lack of virtually any gravitational gradient between the surgical wound and the right ventricle, as well as the high central venous pressure caused by the prone position, it seems that air embolisms constitute a major etiology of CAs during spinal surgery.[11] One case specifically mentions oxygen produced from hydrogen peroxide used for wound irrigation as the cause of an air embolism.[17] Air embolisms are in particular associated with the sitting position, but it is likely that these are undersuspected and underreported as the cause of serious complications in spine surgery.[20]

Extensive blood loss is not uncommon, particularly during deformity correction procedures. As aortic laceration can occur even in minor procedures such as microdiscectomy, and the number of blood transfusions correlates well with the risk for CA, hypovolemia represents a relevant etiology of CA in spine surgery.[4,14] The use of cell savers and low-dose tranexamic acid in high-risk spine surgery can reduce transfusion-related risk.[27]

There have been several reports of cardiac complications coinciding with dural traction or stimulation. Vagal stimulation and brainstem shift mediated by dural manipulation have been hypothesized as mechanisms. Such events are reported regularly in cervical spine surgery, but the incidence seems to be lower during lumbar procedures.[10] Two reports mention episodes of severe bradycardia directly after incidental durotomy, even leading to transient asystole.[9,10] Electrical stimulation of parasympathetic nerve endings with the use of electrocautery can also lead to severe bradycardia.[13] Typically, these reflex-mediated cardiac complications are terminated by eliminating the surgical stimulus, and they do not regularly require CPR.[4,28]

The prone position itself is associated with a variety of complications because of pressure on anterior structures, which leads to hemodynamic changes and difficulties in airway management owing to tracheal compression.[29] Specific complications resulting from compression of anterior structures include pressure sores, postoperative vision loss, lateral femoral cutaneous nerve neuropathy, and abdominal organ failure owing to prolonged ischemia.[30] The prone position requires specific patient positioning that can sometimes lead to vessel occlusion and reduced venous return. This becomes problematic during unexpected blood loss and hypovolemia, which in combination with reduced venous return can quickly escalate to reduced cardiac output.[29]

Genetic Risk Factors

Although ventricular fibrillation or sudden cardiac death at a relatively young age is uncommon, an important subset has a genetic background, and identifying this background has important implications for first-degree family members. Inherited channelopathies and cardiomyopathies should be included in the differential diagnosis.[8,31] Cases such as the one we describe need careful cardiologic and genetic evaluation to identify an underlying cause. In patients with structurally normal hearts, the underlying diseases such as CPVT, Brugada syndrome, long QT syndrome, and short QT syndrome should be considered. A detailed explanation of these entities is provided in **Supplementary Material 1**.[7,8,31,32]

Initial Management

If intraoperative CA is suspected and subsequently confirmed, it is essential to start basic life support as soon as possible, following the guidelines of the American Heart Association or European Resuscitation Council.[6,33] Whenever possible, the lead should be given to an experienced

anesthesiologist or intensive care specialist. However, the neurosurgeon still has an important role in delivering care, in this case to rotate the patient safely from the prone position. We will not discuss the general principles of intraoperative CPR and advanced cardiac life support, but instead focus on the specific challenges that spinal neurosurgery and the prone position pose. **Figure 3** provides a detailed recommendation of the initial steps that neurosurgeons should take when intraoperative cardiac arrest is suspected.[6,33,34]

After CA has been confirmed and the team has been informed, any surgical causes must be eliminated. The surgical procedures should be stopped as soon as possible; this means that hemostasis should be achieved, and that the surgical wound should be protected against trauma and contamination. The evidence for prone resuscitation is weak.[30] Thus, whenever possible, the patient should be immediately turned into supine position as this enables better tracheal access and facilitates effective CPR (**Figure 3**). Instrumentation should be removed as soon as possible. Tamponade, temporary wound closure with a rescue suture, and a temporary sterile covering should be considered before turning the patient. In cervical spine surgery, where a Mayfield clamp is used and where the cervical spine may be unstable intraoperatively, a stiff neck brace should be attached as soon as possible to prevent damage to the spinal cord during CPR.

If venous air embolism is suspected, the patient's head should stay down to increase venous pressure, and the surgical field should be flooded with saline. Echocardiography should be done in cases of suspected venous air embolism to find evidence of increased right ventricular load, and patients may require central venous cannulation. Additional causes, such as major hemorrhage must be identified and treated according to protocol.[35]

CONCLUSION

Common surgical causes such as venous air embolism, excessive hemorrhage, dural traction or stimulation, and anaphylactic shock should be considered. In patients with structurally normal hearts, rhythm abnormalities that can be provoked by adrenergic stimulation owing to rarer genetic etiologies have to be included in the differential diagnosis. In cardiac arrests associated with spine surgery in the prone position, patients should be rotated for standard supine CPR, whenever possible.

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SUPPLEMENTARY MATERIAL 1

GENETIC ETIOLOGIES OF CARDIAC ARREST IN PATIENTS WITH STRUCTURALLY NORMAL HEARTS

Catecholaminergic Polymorphic Ventricular Tachycardia

A resting electrocardiogram may be unremarkable. Exercise, stress, or emotion, such as during a surgical intervention, can trigger reproducible ventricular arrhythmias. Approximately 79% of untreated individuals with CPVT experience cardiac events, and the incidence of sudden cardiac death (SCD) is 30% among this population.[1] Mutations in *RyR2* and *CASQ2*, the main genes underlying CPVT, can be identified in up to 60% of patients. If an exercise test is not possible for initial evaluation, as in our patient, an intravenous epinephrine stress test is diagnostically valuable and has high specificity for the disease.[2,3] Antiadrenergic therapy with β-blockers is effective.

Brugada Syndrome

Patients with Brugada syndrome (BrS) show "coved" ST segment elevation in right precordial leads and right bundle-branch blocks. Arrhythmias in patients with BrS usually occur at rest, and they can be triggered by fever. Several genes—including *SCN5A*, *GPD1-L*, *SCN1B*, *SCN3B*, *CACNA1C*, and *CACNB2*—are associated with BrS, although only up to 30% of patients have identifiable disease-causing mutations, mainly in *SCN5A*.[1] As medical therapies are ineffective in reducing SCD, the use of implantable cardioverter-defibrillators (ICDs) should be considered in the high-risk population.

Long QT Syndrome

Patients may exhibit QT prolongation, peculiar ST T-wave morphology, and syncope. Given incomplete penetrance and QT variability in the normal population, serial electrocardiograms, Holter monitoring, exercise stress testing, or pharmacologic challenge can be diagnostically useful. Linked genes commonly include *KCNQ1*, *KCNH2*, and *SCN5A*. Severely prolonged QT intervals, recent syncope, and *SCN5A* mutations are associated with a higher risk of cardiac events. In addition to avoiding QT prolonging medication, initial antiadrenergic therapy with β-blockers is recommended, and ICD implantation or left cardiac sympathetic denervation can be considered in highly symptomatic patients.[1]

Short QT Syndrome

Typically, a shortened QT interval, arrhythmias, and SCD occur in these patients. Short QT syndrome is a rare disease, with multiple associated genetic mutations in *KCNH2*, *KCNQ1*, and *KCNJ2* genes. As medical therapy is largely ineffective, ICD implantation is recommended.[1]

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