Egyptian Journal of Anaesthesia

ISSN: (Print) 1110-1849 (Online) Journal homepage: https://www.tandfonline.com/loi/teja20

General anaesthesia versus combined spinal epidural anaesthesia in the presence of mild to moderate pericardial effusion: A study of volunteers undergoing caesarean section

Ahmed S. Elgebaly & Tark Elhawary

To cite this article: Ahmed S. Elgebaly & Tark Elhawary (2013) General anaesthesia versus combined spinal epidural anaesthesia in the presence of mild to moderate pericardial effusion: A study of volunteers undergoing caesarean section, Egyptian Journal of Anaesthesia, 29:1, 19-24, DOI: 10.1016/j.egja.2012.08.005

To link to this article: https://doi.org/10.1016/j.egja.2012.08.005

Egyptian Journal of Anaesthesia (2013) **29**, 19–24

Egyptian Society of Anesthesiologists

Egyptian Journal of Anaesthesia

www.elsevier.com/locate/egja
www.sciencedirect.com

Research Article

General anaesthesia versus combined spinal epidural anaesthesia in the presence of mild to moderate pericardial effusion: A study of volunteers undergoing caesarean section

Ahmed S. Elgebaly *, **Tark Elhawary**

Department of Anesthesia and SICUD, Faculty of Medicine, Tanta University, Egypt
Department of Obstetrics and Gynecology, Faculty of Medicine, Tanta University, Egypt

Received 16 May 2012; revised 30 August 2012; accepted 30 August 2012
Available online 16 November 2012

KEYWORDS

General;
Combined spinal epidural;
Pericardial effusion;
Caesarean section

Abstract *Study objective:* This study evaluated the haemodynamic effects of general anaesthesia versus combined spinal epidural anaesthesia in patients undergoing caesarean section in the presence of mild to moderate pericardial effusion.
Design: A prospective randomized study.
Setting: The study setting included a hospital where a surgical team performed elective caesarean section in the presence of mild to moderate pericardial effusion.
Patients and interventions: Thirty healthy patients were randomly divided into two groups, general anaesthesia (GA) (group I) and combined spinal epidural (CSE) anaesthesia (group II).
Measurements and main results: Heart rate, central venous pressure, mean arterial blood pressure, and pulmonary capillary wedge pressure were measured 10 min before anaesthesia, after 20 and 30 min of anaesthesia, and 30 min after recovery. Blood loss was significantly lower in group II [465.33 (72.78) ml] as compared to group I [548.20 (22.73) ml]. The pain score in group II was significantly lesser [1.66 (0.72)], than in group I [2.60 (0.73)]. The HR was significantly higher in group I as compared to that in group II at 20 and 30 min after anaesthesia, and 30 min after surgery, being 81.53 (2.72), 94.80 (3.12) and 82.8 (2.85) (beats/min), respectively. However, the CVP was significantly higher in the group I at 20 and 30 min after induction, being 8.40 (0.63) and 7.80 (0.67) (cmH2O) respectively. The MAP was significantly higher in group II than in group I at 20 and 30 min after induction, being 80.86 (1.30) and 81.00 (1.00) (mmHg) respectively. The PCWA was

* Address: Department of Anesthesia and SICUD, 43 IBN Elfard Street, Elgharbia, Tanta, Egypt. Tel.: +20 01005121944.
E-mail address: elgebaly_13@hotmail.com (A.S. Elgebaly)
Peer review under responsibility of Egyptian Society of Anesthesiologists.

Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.egja.2012.08.005

significantly higher in group I compared to group II at 20 and 30 min after induction, being 10.13 (1.35) and 11.80 (0.94) (mmHg), respectively.

Conclusion: CSE anaesthesia appeared to be more advantageous, in patients undergoing caesarean section with mild to moderate pericardial effusion, with less haemodynamic changes, decreased blood loss, and better postoperative analgesia than general anaesthesia patients.

1. Introduction

Pericardial effusion in pregnancy is a clinically silent entity as neither pericardial pain nor pericardial friction rub can be observed and it appears typically transiently late in pregnancy after the 32nd week, and cannot be detected at 2 months after delivery [1]. The etiology of pericardial disease causing pericardial effusion in pregnant women is the same as in non-pregnant women [2]. There is an excessive water and salt retention in women, hence a significantly higher mean weight gain in late gestation. Patients who gain more than 12 kg during pregnancy are reported to have pericardial effusion than those who gain less than 12 kg. In a cohort study, primigravidas appeared to be more likely to have effusions than multigravidas, with an incidence of 9/13 (69.2%) versus 14/39 (35.9%) [3]. Pericardial effusions are classified as large, moderate, and small based on Horowitz's criteria [4].

Since pericardial effusion cannot be detected by clinical examination or ECG, echocardiography affords a recent non-invasive diagnostic approach [5–7]. The use of echocardiography permits one to estimate as well as to evaluate myocardial performance, chamber size in the basal state and different stages of pregnancy and in the perperium. Rubier et al. [7] has stated that echo-Doppler study is the only technique that can be employed with complete safety, reliable, repeated frequently and entirely without patient discomfort so, the reluctance to utilise X-rays and other invasive diagnostic tools during pregnancy which has been a limiting factor in the ability to differentiate between normal physiologic changes and organic heart disease [5,6].

Prevention of hemodynamic instability during caesarean delivery during anaesthesia has been the aim of several studies [8–10]. Non-invasive monitoring has been used in all previous studies. But we used continuous invasive monitoring in pregnant women undergoing caesarean section with mild to moderate pericardial effusion. Hemodynamic monitoring with a balloon flotation pulmonary artery catheter is useful in mild to moderate effusion as it helps to avoid hemodynamic complications and aids in fluid and inotropic resuscitation [10]. The aim of this randomized trial was to investigate the effects of mild to moderate pericardial effusion on the suitability of Combined Spinal Epidural (CSE) using a double interspace technique in patients undergoing caesarean. CSE is associated with the advantages of decreased liability for cardiac decompensation, decreased blood loss, lower failure rate, better postoperative pain scores, lower incidence of hypotensive episodes, and lower maternal and umbilical cord blood concentrations of local anaesthetics [10].

2. Patients and methods

Study approval was obtained from the Institutional Ethics and Research Committee and informed written consent was obtained from all patients included in this study, which was performed in the Obstetric and Gynaecology Department. 117 pregnant women of ASA grade I/II, with a mean age of 35 (25–40) years were selected during a 2 year period. They were examined systematically and clinically and were investigated by echocardiography on the 12th, 24th, and 34th weeks of gestation, and on the 8th week after delivery.

Echocardiography was performed on the left lateral position using the M-mode and two dimensional modalities in the parasternal long axis, short axis and apical four-chamber views utilising SONO S1000 (Hewlett Packard Ultrasound Imaging System) as well as ATL MK700 System with 2.5 MHz transducer.

Echocardiographically, pericardial effusion was defined by the presence of persistent posterior echo-free space throughout the cardiac cycle, and separating the epicardium from the pericardium associated with flattening of the pericardial echo relative to the epicardial echo. This was obvious and clear in the short axis plane of the left ventricle. Specific attention was focused on the echocardiographic anatomy of the pericardial space and the distribution of different sized effusions. The ellipsoid formula was used to measure the pericardial volume which is the difference between the two volumes (the pericardial sac volume and the cardiac volume [6]).

Pericardial effusion was classified as a mild (pattern C with < 10 mm), moderate (pattern D with 10–20 mm), and large effusion (pattern D with > 20 mm) based on Horowitz's criteria described by Weitzman et al. [9] and Horowitz et al. [11]. All participants included in the study were free of any cardiac problems; pregnancy was within normal limits according to obstetric criteria in all instances at 37th weeks of gestation to avoid the stress of labour and vaginal delivery and with mild to moderate pericardial effusion. Pregnant women undergoing emergency surgery, pericardial rub or any sign of pericardial involvement, sinus tachycardia (> 100 beats/min), with allergies to any of the drugs used during anaesthesia or with large effusion were excluded from the study.

Peripheral wide bored venous canulae were placed in all patients before anaesthesia induction; the heart rate and non invasive mean arterial blood pressure recorded served as baseline observations for subsequent comparison. A Swan-Ganz catheter was inserted in patients of the (GA) group immediately after endotracheal intubation or after local anaesthesia and 1 μm/kg fenatyl for (CSE) group. In addition, we performed electrocardiography and pulse oximetry, and continuously monitored pulmonary artery pressure, end tidal carbon dioxide, anaesthetic agent concentrations, and hourly urine output in all patients. The patients were randomly divided into general anaesthesia (GA) group combined spinal epidural (CSE) group by the sealed envelopes method [12]. The anesthesiologist responsible for the further evaluation of the patient of both groups was unaware of the group allocated and replaced

the colleague who had performed the anaesthesia. Heart rate, central venous pressure, mean arterial blood pressure, and pulmonary capillary wedge pressure were measured10 min before anaesthesia, after 20 and 30 min of anaesthesia, and 30 min after recovery.

In the general anaesthesia (GA) group, preoxygenation was administered with 10 L/min for at least 3 min, followed by intravenous induction of anaesthesia using ketamine at a dose of 2 mg/kg, followed by 2 mg/kg suxamethonium. Cricoid pressure was applied as consciousness was lost and maintained until tracheal intubation and cuff inflation was performed and confirmed to be leak free. Further, 50% nitrous oxide: 50% oxygen was administered with 0.75% isoflurane which was continued until the end of the procedure.

Muscle relaxation was achieved with 0.15 mg/kg of pancuronium after the suxamethonium was weaned off, and a peripheral nerve stimulator was used to assess the neuromuscular blockade. Following delivery of the baby, the anaesthesia was deepened with 0.1 mg/kg morphine i.v., reducing the inspired oxygen concentration to 33%, and removing the wedge. Finally, any residual neuromuscular block was reversed by neostigmine 0.08 mg/kg and atropine 0.02 mg/kg, and the patient was given 100% oxygen, extubated lying sideways, and kept awake after thorough suction of the pharynx. The risks of aspiration were reduced by emptying the stomach before extubation. Patients were moved to the recovery area where the pain score was assessed at 30 min after surgery using a visual analogue scale (VAS) (0 = no pain; 10 = most severe pain), and analgesia was simultaneously administered with patient control analgesia(PCA) in a dose of 2 mg with a lockout of 6 min for 30 min postoperative.

The CSE group was given anaesthesia via a combined spinal epidural (CSE) using the double interspace technique. Under aseptic precautions, with the patient in the sitting position, an epidural catheter was inserted in the L2–L3 interspace. Subsequently, a subarachnoid block with 7 mg of 0.5% hyperbaric bupivacaine with 20 mcg of fentanyl [8,13] (total volume, 1.8 mL) was administered in the lower interspace. The sensory level was achieved at T8. Further, 3 mL of 2% lignocaine with 15 μg adrenaline was administered through the epidural catheter to achieve a T6 level. Oxygen at 4 L/min was administered, and left uterine displacement of 15° was maintained throughout the procedure. Postoperatively, patients were shifted to the PACU with the epidural catheter in situ. For postoperative pain relief analgesia was simultaneously administered using PCA under intensive monitoring and VAS scores were assessed, and. Subsequently, the catheter was removed.

For both groups, following the delivery of the baby, a slow oxytocin drip (20 units in 500 mL normal saline at 100 mL/h) was started. The average volume of total fluids received was 500 mL crystalloid, and the mean urine output was 400 mL.

3. Statistical analysis

Sample size was calculated from a power analysis with a two-sided *t*-test based on data from previous studies [1,3–6] which showed significantly, pericardial effusion of variable volume but always asymptomatic and latent was observed in 40.1 per 100 of patients at the end of pregnancy on echocardiographic examination. Considering potential loss of data, we decided to recruit 117 parturients. A minimum of 15 subjects in each group was calculated as being necessary to demonstrate a difference, with a power greater than 80% and at a significance level of 0.05 Statistical analysis was performed using the unpaired Student's *t*-test or rank-sum test, and the chi-square or Fisher's exact test, as appropriate. Significance was indicated by $P < 0.05$.

4. Results

Using the Horowitz criteria, pericardial effusion was detected by M-mode and cross-sectional echocardiography in 37 cases. The effusion was found to be large in seven cases using the ellipsoid formula and these cases were excluded from the study. Statistical analysis was performed in the remaining 30 (10 moderate and 20 mild cases), which were further divided into 15 cases in each group. Eight weeks after delivery, echocardiographic studies did not show any evidence of pericardial effusion in 17 participants, but there was major reduction in the posterior echo-free space in 20 participants.

No significant differences were observed between the two groups with respect to age, height, weight, or duration of study and monitoring. Intraoperative blood loss was significantly lower in group II (CSE group) than in group I (GA group) (Table 1).

The immediate (30 min) postoperative pain score and morphine consumption (mg) in group II was 1.66 (0.72) and 4.2 (1.3), which was significantly lower than that in group I [2.60 (0.73)] and [7.4 (2.2)] (Table 2).

Table 3 shows the haemodynamic values before, during, and after induction of anaesthesia and surgery. No significant differences were noted between the two groups for the heart rate, MAP, CVP or PCWP 10 min before the induction of anaesthesia.

The HR was significantly higher in group I as compared to that in group II at 20 and 30 min after anaesthesia, and 30 min

Table 1 Demographic data and intraoperative blood loss of the study patients in both groups. Values are means (SD).

	Group I (GA) ($n = 15$)	Group II (CSE) ($n = 15$)
Age (years)	42.66 (3.97)	41.13 (4.48)
Height (cm)	171.53 (2.29)	170.33 (2.05)
Weight (kg)	83.73 (2.84)	88.33 (6.34)
Duration of study and monitoring (min)	115.27 (5.45)	112.87 (4.58)
Total blood loss (ml)	548.20 (22.73)	465.33 (72.78)*

* Indicates $p < 0.05$ (statistically significant) when compared with the other group.

Table 2 Pain scores and Morphine consumption of the study groups. Values are means (SD).

	Group I (GA) ($n = 15$)	Group II (CSE) ($n = 15$)
30 min postoperative pain score	2.60 (0.73)	1.66 (0.72)*
30 min post operative Morphine consumption (mg)	7.4 (2.2)	4.2 (1.3)*

* Indicates $p < 0.05$ (statistically significant) compared to other group.

Table 3 Haemodynamics data for patients in the two groups of the study. Values are presented as mean (SD).

	Parameter	10 min before induction of anesthesia	20 min after induction of anesthesia	30 min after induction of anesthesia	End of surgery	30 min after end of surgery
Group 1 (GA) (n = 15)	Heart rate	79.20 (3.00)	97.13 (4.08)*	98.20 (3.87)*	89.73 (3.61)	86.00 (2.75)*
	CVP	9.80 (1.08)	8.40 (0.63)*	7.80 (0.67)*	7.80 (0.67)	7.33 (0.72)*
	MAP	80.66 (1.11)	74.86 (2.13)	74.3 (2.38)	71.93 (1.03)	71.93 (1.03)
	PCWP	11.46 (0.83)	10.13 (1.35)*	11.80 (0.94)*	8.66 (0.61)	9.66 (1.17)
Group 2 (CSE) (n = 15)	Heart rate	79.20 (3.16)	81.53 (2.72)	94.80 (3.12)	90.40 (3.45)	82.8 (2.85)
	CVP	9.53 (0.91)	7.20 (0.56)	6.26 (0.59)	7.53 (0.83)	6.06 (0.59)
	MAP	80.53 (0.83)	80.86 (1.30)*	81.00 (1.00)*	71.20 (1.14)	72.13 (1.18)
	PCWP	11.40 (0.50)	5.86 (0.63)	6.93 (0.59)	8.40 (0.98)	9.20 (0.67)

HR = heart rate (beats/min); CVP = central venous pressure (cmH2O); MAP = mean arterial blood pressure (mmHg); PCWP = pulmonary capillary wedge pressure (mmHg).
* Denotes $p < 0.05$ compared with the other group.

after surgery, being 81.53 (2.72), 94.80 (3.12) and 82.8 (2.85) (beats/min), respectively. However, the CVP was significantly higher in the group I at 20 and 30 min after induction, being 8.40 (0.63) and 7.80 (0.67) (cmH2O) respectively. The MAP was significantly higher in group II than in group I at 20 and 30 min after induction, being 80.86 (1.30) and 81.00 (1.00) (mmHg) respectively. The PCWA was significantly higher in group I compared to group II at 20 and 30 min after induction, being 10.13 (1.35) and 11.80 (0.94) (mmHg), respectively.

5. Discussion

Although the true incidence of pericardial disease in pregnancy remains unknown, many pregnant women are known to develop minimal to moderate pericardial effusion by the third trimester of pregnancy; this process tends to be transient and clinically silent [1]. Low voltage is one of the ECG manifestations of pericardial effusion, but it is not specific or sensitive enough to diagnose the presence of pericardial effusion and most of these effusions disappear within a month postpartum and are simply reflected as an incidental findings in echocardiography [3].

Abduljabbar et al. [3] evaluated 52 normal pregnant women by serial echocardiography during their first, second, and third trimesters; asymptomatic effusions were found to be relatively common as follows. Eight women (15.4%) showed effusion on echocardiography in the first trimester, while in 10 (19.2%) and 23 (44.2%) women, effusions were noted in the second and third trimesters, respectively. Haiat et al. [1,4] performed serial M-mode and 2-dimensional echocardiography at various stages of gestation in 123 asymptomatic gravida women. Of these, 19 (15.4%) showed unexpected signs of pericardial effusion on the echocardiogram. The effusions were categorised as large in 2, moderate in 4, and small in 13. Apart from the signs of pericardial effusion, the echocardiograms in 16 of the 19 women appeared normal Haemodynamic abnormalities, caused by pericardial effusion, range from undetectable or mild, to life threatening. The pericardium is scarred and thus adds an element of constrictive pericarditis. Pericardial adhesions or organisation of the fluid can result in localised, and thus atypical and tamponade, which are the causes of a number of distinct clinical and haemodynamic syndromes [14–16].

Pericardial effusions and tamponade are often obscured by the haemodynamic changes that occur in normal pregnancy. Sinus tachycardia, ventricular premature beats, and ECG changes of pericarditis are sometimes difficult to discern from slight ST-segment depressions and T-wave changes that are typically observed in normal pregnancy. Pulsus paradoxus, which is helpful in diagnosing cardiac tamponade, can also be noted in normal late pregnancy even in the absence of pericardial effusion, as well as in approximately 50% of patients with chronic constrictive pericarditis, chronic obstructive lung disease, pulmonary embolus, obesity, and hypovolemic shock [4].

The infrequency of effusive and compressive pericardial disease limits the feasibility of large, randomized studies to compare the effectiveness of different anaesthetic strategies in patients undergoing caesarean sections. The present study was designed to compare the haemodynamic effects of GA and CSE anaesthesia in patients undergoing caesarean section diagnosed with pericardial effusion. We accordingly evaluated the incidence of tachycardia, hypotension, and increased central venous pressure and pulmonary capillary wedge pressure with increased liability for cardiac decompensation. The present study evaluated the efficacy of a combined spinal epidural anaesthesia, which was assumed to be identical to those of epidural anaesthesia with the added benefit of rapid onset due to the spinal component. A low dose of local anaesthetic in the spinal component can be used, with later extension with the epidural if necessary. Further, the advantages of CSE over epidural anaesthesia alone include a lower failure rate, better postoperative pain scores, lower incidence of hypotensive episodes, and lower maternal and umbilical cord blood concentrations of local anaesthetics [13].

Our findings revealed that CSE anaesthesia is a safe alternative anaesthetic technique in patients undergoing caesarean sections with mild to moderate pericardial effusion for the advantageous in of lower intraoperative blood loss, lesser immediate postoperative VAS pain score and more haemodynamic stability compared to GA.

Disadvantages of GA technique that agree with the present study results have been reported previously, suggesting that while GA may be used for emergent caesarean sections, its use is associated with severe tachycardia and hypotension in patients with pericardial effusion [14,15]. This was well documented by Murray and Robertson [14] in two patients with unsuspected tamponade who were anaesthetised with low concentrations of nitrous oxide and halothane. The resulting

hypotension in both these patients was unresponsive to rapid intravenous administration of fluids, termination of anaesthesia, and the administration of typical vasopressors. It was only corrected when the pericardial effusions were relieved. Similarly, hypotension and cardiac arrest have been reported in patients with tamponade after the administration of thiopental, d-tubocurarine, atropine, most general anaesthetics, and even preoperative sedations [16]. All inhalation and most intravenous anaesthetics are known to decrease myocardial contractility [15].

Moreover, cyclopropane, ether, and possibly ketamine are peripheral vasodilators, except when used at low concentrations [17,18]. Although agents that maintain vascular tone and venous return by stimulating the release or preventing the uptake of norepinephrine would not cause these effects [7,8], since a patient with cardiac tamponade may be under the effects of maximum endogenous catecholamine stimulation before the induction of anaesthesia, even such agents may result in vasodilatation and a reduced venous return.

Arrhythmias are common in patients with tamponade [7,8]. In these patients, even minimal hypoxia and hypercarbia can trigger arrhythmias [15]. Intermittent positive-pressure breathing may cause further tamponade of the heart, lungs, and intrathoracic great veins. Similar to the findings of present study, Ammar et al. [19] previously suggested that controlled ventilation may be a major cause of hypotension in an anaesthetised patient with tamponade. However, such ventilation is an essential component of the general anaesthesia technique in these patients in order to prevent hypoxia and hypercarbia.

As indicated in the present study, several authors have previously suggested the use of ketamine for analgesia or anaesthesia because ketamine causes less respiratory depression and tends to support heart rate, contractility, and systemic vascular tone better as compared to other drugs [25,20,21]. Spontaneous ventilation and intravascular volume expansion may help in maintaining and enhancing venous return and subsequently improve ventricular filling pressures [25,22].

Our findings revealed that HR, CVP and PCWP seems to be significantly higher in group I (GA group) than group II (CSE group) which agreed with the results of Teoh and Sia [13] and explained as the stress of laryngoscopy and intubation coupled with the cardiodepressant effects of general anaesthetics which explain significantly higher MAP in group II than in group I at 20 and 30 min after induction thus points towards the need for an alternative, i.e. a regional anaesthetic technique.

While vasodilatation produced by regional anaesthesia is beneficial in reducing the after-load and improving the forward flow, spinal anaesthesia can result in a precipitous fall in blood pressure that can be detrimental in a patient with already compromised cardiac function and this explain the underlying fact at our study that the CVP and PCWP were lower in the CSE group may be simply interpreted as a physiological haemodynamic consequence of the sympatholysis and vasodilatation associated with epidural anaesthesia. On the contrary our findings revealed that The MAP was significantly higher in group II than in group I at 20 and 30 min after induction which reflected from decreasing MAP in group I and stability of MAP in group II.

In our study the use of invasive monitoring made it possible to detect the immediate hemodynamic changes after CSE anaesthesia. The vasodilatory effect of spinal anesthesia is expected, but the immediate effect on SVR and CO has been invasively continuously monitored. These changes are of clinical relevance regarding regional anaesthesia to pregnant women at risk (i.e., cardiac disease) where prominent hemodynamic changes could be harmful and should be prevented. Similar to our procedure Langesaeter et al. [23]. Gambling et al. [24] described that the haemodynamic state should be optimised by careful fluid replacement under the control of invasive monitoring prior to the institution of a regional block. Since our subjects were relatively asymptomatic and remained haemodynamically stable throughout pregnancy, we chose to monitor central venous pressure and pulmonary capillary wedge pressure. However, similar cases have been previously managed successfully with non-invasive monitoring alone [25].

In cases of symptomatic pericardial effusion, Breen and his colleague Janzen [25] preferred catheterisation of the right heart using a Swan-Ganz catheter, which may be necessary to document cardiac tamponade and/or to exclude constriction. However, Shibli et al. [26] used left heart catheterisation with appropriate shielding, indicating that this may be necessary for confirming the diagnosis of constriction or aortic dissection, particularly if surgery is planned. For such symptomatic patients, the brachial approach may be preferable to avoid foetal exposure to radiation.

Our study has an important limitation of small sample size. The study included only 30 participants who fulfilled all the inclusion criteria and had undergone caesarean section in the presence of mild to moderate pericardial effusion. The sample size was restricted to 30cases because of logistical reasons; Swan-Ganze was provided free of cost to the study participants, limiting the inclusion of more cases. Another limitation was the absence of images in this study, we thought that it will not add relevant information for the reader, as the article contains many Refs. [5–7,17] which include a good (photo) images and detailed description for the types effusion.

6. Conclusion

This prospective, randomised study was designed to evaluate and assess the haemodynamic effects of GA versus CSE anaesthesia in patients undergoing caesarean section diagnosed with pericardial effusion and to clarify the advantages of CSE over GA in such patients. Based on our findings of significant low HR, CVP and PCWP and significant high MAP, further randomised controlled trials are needed, to consider CSE to be a safe and acceptable option for patients with pericardial effusion undergoing caesarean section, with preoperative evaluation and invasive haemodynamic monitoring are the key determinants of successful outcome.

Acknowledgments

We would like to thank Dr. Timor Mostafa Abdalla, MD. Department of Cardiology, Faculty of Medicine, Tanta University, Egypt, for his assistance in using TTE in this study. Also, we thank all of the nurses in our postanaesthesia care unit for caring our patients.

References

[1] Haiat R, Halphen C, Clément F, Michelon B. Silent pericardial effusion in late pregnancy. Chest 1981;79(6):717.

[2] Azimi NA, Selter JG, Abott JD. Angiosarcoma in a pregnant woman presenting with pericardial tamponade: a case report and review of the literature. Angiology 2006;57(2):251–7.
[3] Abduljabbar HS, Marzouki KM, Zawawi TH, Khan AS. Pericardial effusion in normal pregnant women. Acta Obstet Gynecol Scand 1991;70(4–5):291–4.
[4] Haiat R, Halphen C. Silent pericardial effusion in late pregnancy: a new entity. Cardiovasc Inter Radiol 1984;7(6):267–9.
[5] Halphen C, Haïat R. Echocardiographic detection of asymptomatic pericardial effusion during normal pregnancy. Arch Mal Coeur Vaiss 1983;76(1):71–6.
[6] Mohamed SM, Mohmmed RA, Abdulkader RA, Mostafaal-shamiri , Randa MN, Zainab AB, Hussain AY. Non-invasive evaluation of cardiac changes in normal pregnant women, their clinical significance and implications. J Saudi Heart Assoc 2001;13(1):20–5.
[7] Rubier S, Damani PM, Pinto ER. Cardiac size, performance estimated with echocardiography. Am J Cardiol 1977;40:534–40.
[8] Chaudhary S, Salhotra R. Subarachnoid block for caesarean section in severe preeclampsia. J Anaesthesiol Clin Pharmacol 2011;27:169–73.
[9] Weitzman LB, Tinker WP, Kronzon I. The incidence and natural history of pericardial effusion after cardiac surgery: an echocardiographic study. Circulation 1984;69:506–11.
[10] Shabetai R. Pericardial effusion: haemodynamic spectrum. Heart 2004;90(3):255–6.
[11] Horowitz MS, Shulz CS, Stinson EB. Sensitivity and specificity of echocardiographic diagnosis of pericardial effusion. Circulation 1974;50:239–47.
[12] Pocock SJ. Method of randomization. In: Pocock SJ, editor. Clinical trials. Apractical approach. New York: John Wiley and Sons; 1994. p. 66–89.
[13] Teoh WH, Sia AT. Ultra-low dose combined spinal-epidural anaesthesia for caesarean section in severe pre-eclampsia. Anaesthesia 2006;61:511–2.
[14] Murray BRP, Robertson DS. Anaesthesia for mitral valvulotomy complicated by hypotension due to pericardial effusion. Brit J Anaesth 1964;36:256–8.
[15] Moon GJ. Pericardial effusion and severe tachycardia. Anes Analg 1982;61:38.
[16] Mohamed SM, Mohmmed RA, Abdulkader RA, Mostafaal-shamiri , Randa MN, Zainab AB. Screening and localization of silent pericardial effusion in healthy pregnant women. J Saudi Heart Assoc 2001;13(2):113–6.
[17] Eger EI, Smith NT, Cullen DJ. A comparison of the cardiovascular effects of halothane, fluroxene, ether and cyclopropane in man. Anesthesiology 1971;34:25–41.
[18] Stanley TH, Weidauer HE. Anesthesia for the patient with cardiac tamponade. Anasth Analg 1973;52:110–4.
[19] Ammar T, Reich DL, Kaplan JA. Uncommon cardiac diseases. In: Benumof JL, editor. Anesthesia and uncommon diseases. Philadelphia: WB Saunders Co.; 1998. p. 70–122.
[20] Lake CL. Anesthesia and pericardial disease. Anesth Analg 1983;62:143–431.
[21] Stoelting RK. Nonbarbiturate induction drugs. In: Stoelting R, editor. Pharmacology and physiology in anesthetic practice. Philadelphia, Pennsylvania: Lippincott; 1999. p. 104–57.
[22] Stoelting RK, Dierdorf SF. Pericardial diseases. In: Stoelting RK, Dierdorf SF, editors. Anesthesia and co-existing disease. New York: Churchill Livingstone Inc.; 2002. p. 135–42.
[23] Langesaeter E, Rosseland LA, Stubhaug A. Continuous invasive blood pressure and cardiac output monitoring during cesarean delivery: a randomized, double-blind comparison of low-dose versus high-dose spinal anesthesia with intravenous phenylephrine or placebo infusion. Anesthesiology 2008;109(5):856–63.
[24] Gambling DR, Flanagan ML, Huckell VF. Anaesthetic management and non-invasive monitoring for Cesarean section in a patient with cardiomyopathy. Can J Anesth 1987;34:505–8.
[25] Breen TW, Janzen JA. Pulmonary hypertension and cardiomyopathy: anaesthetic management for Caesarean section. Can J Anesth 1991;38:895–9.
[26] Shibli K, Russell I. A survey of anaesthetic techniques used for caesarean section in the UK in 1997. Int J Obstet Anesth 2000;9(3):160–7.