شبكة المعلومات الجامعية

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بسم الله الرحمن الرحيم

HANAA ALY

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جامعة عين شمس

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Role of Minimal Dose of Norepinephrine in Prevention of Hypotension Induced by Subarachnoid Anesthesia in Elective Cesarean Section

Thesis

Submitted for Partial Fulfillment of
Master Degree in **Anesthesia**

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Ahmed Mohamed Kamal

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List of Abbreviations

Abb.	Full term
AV	*Atrioventricular*
CC	*Closing capacity*
CO	*Cardiac output*
CSE	*Combined spinal–epidural*
CSF	*Cerebrospinal fluid*
DOPA	*Dihydroxyphenylalanine*
ECG	*Electrocardiogram*
ERV	*Expiratory reserve volume*
FDA	*Food and Drug Administration*
FRC	*Functional residual capacity*
Hb	*Hemoglobin*
HIV	*Human immunodeficiency virus*
HR	*Heart rate*
IV	*Intravenous*
MAC	*Minimum alveolar concentration*
MHPG	*3-methoxy-4-hydroxyphenylglycol*
NE	*Norepinephrine*
NIBP	*Non-Invasive Blood Pressure*
NS	*Non significant*
PE	*Phenylephrine*
PT	*Prothrombin time*
PTT	*Partial thromboplastin time*
RV	*Residual lung volume*
SD	*Standard deviation*
SPSS	*Statistical Program for Social Science*
SVR	*Systematic vascular resistance*
TEG	*Thromboelastography*
V.C.	*Vertebral Column*
WBC	*White blood cell*

INTRODUCTION

Maternal hypotension is a common complication after spinal anesthesia for cesarean delivery ***(Hasanin et al., 2017)***. Even though there is variability in defining hypotension for expectant mothers involving neuraxial anesthesia, most authors define it as being a 80% reduction in systolic blood pressure, comparing it to initial values (prior to drugs being placed in the neuroaxis) or absolute systolic blood pressure values between 100 mmHg and 90 mmHg ***(Cyna et al., 2006).***

Spinal anesthesia techniques produce hypotension through blockade of sympathetic nerve fibers which control vascular smooth muscle tone. Several studies emphasize that spinal anesthesia induced hypotension is principally related to a marked decrease in systemic vascular resistance rather than decrease in cardiac output ***(McDonald et al., 2011).***

Aortocaval compression caused by mechanical phenomena of the pregnant uterus during the last trimester of pregnancy when a patient adopts a supine position ***(Burns et al., 2001).***

There is a 33% incidence of hypotension caused by spinal block in the general population (non-expectant mothers). This is greater than 90% in pregnant females (depending on the definition used) making this the most frequently occurring adverse effect caused by the intervention described to date.

Multiple pregnancies are not considered to be a risk factor for hypotension caused by spinal anesthesia for caesarean section compared to single pregnancies ***(Reidy and Douglas, 2008).***

Previously, maternal hypotension and fetal outcome were thought to be improved by avoiding aortocaval compression (left uterine displacement) and increasing the blood volume, such as by intravenous fluid loading to increase the venous return, cardiac filling pressure, and cardiac output (CO). These techniques, however, may be in adequate, and use of vasoactive drugs in obstetric patients can be adequately effective in addition to the previous maneuvers for countering the hypotension induced by spinal anesthesia ***(Cyna et al., 2006).***

Vasopressor drugs act on α1-, β1- and β2-adrenoreceptors in the heart and vascular system. The physiological response of these adrenoreceptor agonists depends on the type and location of the receptors. Vasoconstriction is mainly mediated by α1-receptors. However, some vasopressors can also stimulate β1- and/or β2-receptors directly or indirectly, leading to positive inotropic (increasing cardiac contractility) and/or positive chronotropic (increasing heart rate, HR) effects.

The complex hemodynamic effects of the various vasoconstrictors depend on the relative stimulation of these adrenoreceptors. Reflex cardiovascular responses to

vasopressors, on the other hand, may result in other changes, including the unwanted reflex bradycardia ***(Ruy et al., 2019).***

Ephedrine is a sympathomimetic that has both a direct (alpha and beta receptor agonist) and indirect (release of norepinephrine from presynaptic nerve terminals) mechanism of action. Uterine blood flow, in particular was maintained more favorably with beta-agonists than with alpha-agonist ***(Siddik-Sayyid et al., 2014).***

Favorable effects on uteroplacentary circulation can be explained by an increase in nitric oxide synthase and reduced sympathetic innervation of the vascular uterine layer. Ephedrine also presents beta 1 adrenergic action, thereby explaining positive chronotropism and inotropism, thereby substantially increasing heart rate and cardiac load and exercising a modest effect on adrenergic beta 2 receptors. This may partly explain uteroplacentary vasculature dilatation. Its vasopressor action (arterial and venous) is mediated by alpha 1 action ***(Cooper and Mowbray, 2004).***

Norepinephrine is a potent α-adrenergic receptor agonist, it is also a relatively weak agonist at β-adrenergic receptors. This thesis will try to emphasize that norepinephrine might therefore be an effective vasopressor for maintaining blood pressure during spinal anesthesia with less tendency to decrease HR and CO compared with ephedrine. Although treatment of hypotension during spinal anesthesia is listed by the

manufacturer as an indication for the use of norepinephrine, there is limited information available for its use for this purpose in the literature and few reports of its use in obstetric patients ***(Hoyme et al., 2015).***

AIM OF THE WORK

1. The main and primary goal of this research is to evaluate and assess the efficacy of minimal dose of Norepinephrine in controlling the incidence of post spinal hypotension in patients undergoing cesarean delivery.

2. The secondary goal is to evaluate the other associated symptoms related to maternal hypotension e.g. Nausea, Vomiting, Yawing, Maternal PH, Serum lactate level and maternal urine output.

3. Neonatal blood gases and APGAR score.