



**Comparative Study between 25 Gauge Spinal
Needle (Quincke) versus (pencil point) As
Regarding incidence of Post Dural Puncture
Headache**

Thesis

*Submitted for Partial Fulfillment of Master
Degree in Anesthesiology*

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2018***

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سبقتناك يا معلم لنا
إلا ما علمتنا إنك أنت
العليم العظيم

صدق الله العظيم

سورة البقرة الآية: ٣٢

Acknowledgment

*First and foremost, I feel always indebted to **ALLAH**, the Most Kind and Most Merciful.*

*I'd like to express my respectful thanks and profound gratitude to **Prof. Dr. Fahmy Saad Latif Eskander**, Professor of Anesthesia, Intensive Care and Pain Management Faculty of Medicine – Ain Shams University for his keen guidance, kind supervision, valuable advice and continuous encouragement, which made possible the completion of this work.*

*I am also delighted to express my deepest gratitude and thanks to **Dr. Dalia Ahmed Ibrahim**, Lecturer of Anesthesia, Intensive Care and Pain Management Faculty of Medicine – Ain Shams University, for her kind care, continuous supervision, valuable instructions, constant help and great assistance throughout this work.*

*I am deeply thankful to **Dr. Amr Hosni Hamza**, Lecturer of Anesthesia, Intensive Care and Pain Management Faculty of Medicine – Ain Shams University, for his great help, active participation and guidance.*

Mohamed Ehamri Mahmoud

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List of Abbreviations

Abb.	Full term
ADP.....	Accidental dural puncture
ASA.....	American society of anesthesiologists
BMI.....	Body mass index
CNS.....	Central nervous system
CS.....	Caesarean section
CSF	Cerebrospinal fluid
DBP.....	Diastolic blood pressure
EBP	Epidural blood patch
ECG	Electro cardio gram
I.V	Intra venous
I.V.C.....	Inferior vena cava
LP.....	Lumber puncture
PDPH.....	Postdural puncture headache
RCTs	Randomized controlled trials
RR	Relative risk
SBP	Systolic blood pressure
SD	Standard deviation
SpO2	Oxygen saturation

INTRODUCTION

Spinal anesthesia developed in the late 1800s. In 1891, Wynter and Quincke aspirated cerebrospinal fluid (CSF) from the subarachnoid space for the treatment of raised intracranial tension associated with tuberculous meningitis. The catheters and trochars used were probably about 1 mm in diameter and would certainly have led to a post-dural puncture headache (PDPH). However, all Quincke and Wynters' subjects died soon after (*Bleeker et al., 2004*).

In 1895, John Corning, a New York physician specializing in diseases of the mind and nervous system, proposed that local anesthesia of the spinal cord with cocaine may have therapeutic properties. Corning injected cocaine 110 mg at the level of the T11/12 interspace in a man to treat habitual masturbation. Despite being accredited with the first spinal anaesthetic, it is unlikely from his description for the dose of cocaine that his needle entered to the subarachnoid space (*Collier, 2000*).

In August 1898, Karl August Bier, a German surgeon, injected cocaine 10–15 mg into the subarachnoid space of seven patients, himself and his assistant, Hildebrandt. Bier, Hildebrandt and four of the subjects all described the symptoms associated with PDPH. Bier surmised that the headache was attributable to loss of CSF. By the early 1900s, there were numerous reports in the medical literature of the application of

spinal anesthesia using large spinal needles. PDPH was reported to be a complication in 50% of subjects. At that time, the headache was said to resolve within 24 h. Bier also gained first-hand experience of the disabling headache related to dural puncture. He correctly surmised that the headache was related to excessive loss of CSF (*Gogarten and Van Aken, 2000*).

Ether anesthesia was introduced into obstetric practice in 1847, shortly after Morton's public demonstration. Despite the obvious advantages of regional anesthesia for the relief of labour pain, it was not until a Swiss obstetrician in 1901 used intrathecal cocaine for the relief of pain in the second stage of labour that regional anesthesia for obstetrics was popularized. Though both vomiting and a high incidence of PDPH were noted, it was the high mortality rate in Caesarean deliveries performed under spinal anesthesia (1 in 139) that led to the abandonment of this technique in the 1930s. The period from 1930 to 1950 has often been referred to as the 'dark ages of obstetric anesthesia', when natural childbirth and psychoprophylaxis were encouraged (*Webb et al., 2012*).

In 1951, Whitacre and Hart developed the pencil-point needle, based on the observations of Greene in 1926. Developments in needle design since that time have led to a significant reduction in the incidence of PDPH. However, dural puncture headache remains a disabling complication of needle insertion into the subarachnoid space. In the last 50 yr, the development of fine-gauge spinal needles and needle tip

modification has enabled a significant reduction in the incidence of PDPH (*Amorim et al., 2012*).

PDPH is a major complication of neuraxial anesthesia that can occur following spinal anesthesia and with inadvertent dural puncture during epidural anesthesia. Obstetric patients are considered at increased risk for this condition because of their sex, young age, and the widespread use of neuraxial blocks. Inadvertent dural puncture during epidural anesthesia is a more common cause of PDPH than spinal anesthesia because of the use of small, pencil-point needles for spinal anesthesia in this population (*Campbell et al., 1993*).

This is related to how does the microscopic pattern of collagen alignment in the spinal dura affect the dimensions of the dural perforation and How do needle design, size and orientation influence leakage of CSF through the dural perforation and as known that inadvertent dural puncture and PDPH are unavoidable complications so, anesthesiologists need to be familiar with treatment modalities and prevention. This review summarizes current concepts regarding the diagnosis, pathogenesis, pathophysiology, risk factors, prevention, and treatment of PDPH (*Douglas et al., 1997*).

AIM OF THE WORK

The aim of the work is to compare the incidence of post dural puncture headache (PDPH) while using two different spinal needles (quincke) versus (pencil point) both having same gauge (25 gauge) as regarding incidence of PDPH.

Chapter 1

ANATOMY OF DURA MATTER AND PHYSIOLOGY OF CSF

Anatomy of the spinal dura mater

The spinal dura mater is a tube extending from the foramen magnum to the second segment of the sacrum. It contains the spinal cord and nerve roots that pierce it. The dura mater is a dense, connective tissue layer made up of collagen and elastic fibres. The classical description of the spinal dura mater is of collagen fibres running in a longitudinal direction. This had been supported by histological studies of the dura mater. Clinical teaching based upon this view of the dura recommends that a cutting spinal needle be orientated parallel rather than at right angles to these longitudinal dural fibres (*Reina et al., 2000*).

Orientating the needle at right angles to the parallel fibres, it was said would cut more fibres. The cut dural fibres, previously under tension, would then tend to retract and increase the longitudinal dimensions of the dural perforation, increasing the likelihood of a post-spinal headache. Clinical studies had confirmed that PDPH was more likely when the cutting spinal needle was orientated perpendicular to the direction of the dural fibres. However, recent light and electron microscopic studies of human dura mater have contested this

classical description of the anatomy of the dura mater (*Reina et al., 2000*).

These studies describe the dura mater as consisting of collagen fibres arranged in several layers parallel to the surface. Each layer or lamellae consists of both collagen and elastic fibres that do not demonstrate specific orientation. The outer or epidural surface may indeed have dural fibres arranged in a longitudinal direction, but this pattern is not repeated through successive dural layers. Recent measurements of dural thickness have also demonstrated that the posterior dura varies in thickness, and that the thickness of the dura at a particular spinal level is not predictable within an individual or between individuals. Dural perforation in a thick area of dura may be less likely to lead to a CSF leak than a perforation in a thin area, and may explain the unpredictable consequences of a dural perforation (*Eggert and Eggers, 2001*).

Physiology of CSF

CSF is produced primarily in the choroid plexus but there is some evidence of extrachoroidal production. About 500 ml of CSF is produced daily at a rate of approximately 0.35 mL/min and reabsorbed through the arachnoid villa. The total CSF volume in adults is maintained around 150 mL, of which approximately half is extracranial, and gives rise to normal lumbar opening pressures of 5–15 cm H₂O in the horizontal position (40–50 cm H₂O in the upright position) (*Levine and Rapalino, 2001*).

Chapter 2

POST DURAL PUNCTURE HEADACHE

Definition

Headaches following interventions that disrupt meningeal integrity are most commonly labeled (PDPHs). This terminology has been officially adopted in the International Classification of Headache Disorders (*Goldszmidt et al., 2005*).

Incidence

PDPH remains a prominent clinical concern to the present day. Largely due to modifications in practice that followed the identification of risk factors, rates of PDPH following spinal anesthesia have steadily declined, from an incidence exceeding 50% in Bier's time, to around 10% in the 1950s, until currently a rate of 1% or less can be reasonably expected (*Paech et al., 2011*).

However, as perhaps the highest-risk group, an unfortunate 1.7% of obstetric patients continue to experience PDPH after spinal anesthesia using 27-gauge Whitacre needles. Intending to avoid meningeal puncture, epidural techniques are an attractive alternative to spinal anesthesia. Yet, occasional accidental dural puncture ADP, with either the needle or the catheter, is unavoidable (and may be unrecognized at the time

in over 25% of patients who eventually develop PDPH) (*Paech et al., 2011*).

Diagnosis

PDPH presents as a dull throbbing pain with a frontal-occipital distribution. Typically, the headache is aggravated by sitting or standing, and is reduced by lying down. The diagnosis should be questioned in the absence of a postural component of the headache. At least partial relief should occur when the patient assumes the supine position. PDPH may be associated with nausea in >50%, vomiting in <25%, auditory symptoms (tinnitus, hearing loss, hyperacusis) in <15%, visual symptoms (diplopia, photophobia, problems accommodating) in <15% and seizures are rare (*Turnbull and Shepherd, 2003*).

Onset:

According to the International Classification of Headache Disorders criteria for the diagnosis of PDPH, headache develops within 5 days after dural puncture and disappears spontaneously within 1 week, or up to 48 h after an epidural blood patch EBP. The headache may be accompanied by neck stiffness, tinnitus, hypoacusia, photophobia, and nausea. However, recent studies indicate that PDPH occurs within 3 days after dural puncture, and up to 29% of patients have headache as the only symptom (*Amorim et al., 2012*).