# THE STUDY OF UMBILICAL STUMP CHANGES

# A THESIS

SUBMITTED FOR PARTIAL FULFILMENT

FOR THE MASTER DEGREE IN ( OBSTETRICS & GYNAECOLOGY )

19.59 4.8

BY

Hesham Abdel Hay Saleh M.B., B.Ch. 25425

SUPERVISORS

Dr. Sobhi Khalil Abou-Louz

Ass. Prof of Obstetrics & Gynaecology
Faculty of Medicine
Ain Shams University

Dr. Ahmed Abdel Halim El Tawel

Dr. Maher Orman

Prof. of Pathology

Lec. of Obstetrics & Gyngecology

FACULTY OF MEDICINE AIN SHAMS UNIVERSITY

1987

TO MY PARENTS



## **ACKNOWLEDGEMENT**

I would like to express my deep thanks and gratitude to Dr. SOBHI KHALIL ABOU-LOUZ, Assistant Professor of Gynaecology and Obstetrics, Faculty of Medicine Ain Shams University, for giving me the privilege of working under his supervision, for his encouragment, his patience and fruitful guidance throughout the whole work.

Without his continuous support, expert advice, this work have never been complete and come to light.

I would like to express my cordial thanks to Dr. AHHED ABDEL HALIM EL TAWEL, Professor of Pathology, Faculty of Medicine Ain Shams University, for his kindness. He gave me much of his unlimited experience in scientific research which helped me to perform the practical part successfully.

I would like to express my deep thanks to Dr. NAHER OMRAN, Lecturer of Gynaecology of Obstetrics, Faculty of Medicine Ain Shams University, who offered a great help in making this work and for his kind supervision.

My great thanks are offered to the Palestine hospital adminstration and the Microbiology department for their great help to me throughout the whole work.

## **CONTENTS**

|  | PAGE |
|--|------|
| Introduction and Aim of The Work             | 1    |
| Review of Literature                         |      |
| Anatomy                                      | 2    |
| Embryology                                   | 4    |
| Histology                                    | 7    |
| Pathology                                    | 10   |
| Inflammation of Umbilical stumpStaphylococci | 24   |
| Streptococci                                 | 33   |
| Clostridium Tetani                           | 40   |
| Other Causative organisms                    | 43   |
| Complications of umbilical stump sepsis      | 49   |
| Care of umbilical stump                      | 55   |
| Material and Methods                         | 61   |
| Results                                      | 69   |
| Discussion                                   | 77   |
| Summary                                      | 81   |
| References                                   | 84   |
| Arabic Summary .                             |      |

# LIST OF TABLES

|       |     |   | PAGE |
|-------|-----|---|------|
| Table | (1) | : Incidence of staph. aureus and staph. epider-     |      |
|       |     | midis in full-term and premature newborns           |      |
|       |     | in first 6 days of life.                            | 26   |
| Table | (2) | : % of streptococcal colonization, loss of coloniz- |      |
|       |     | ation and acquisition in different groups.          | 35   |
| Table | (3) | : No. of premature neonates colonized with          |      |
|       |     | streptococci.                                       | 38   |
| Table | (4) | : No of cases of neonatal tetanus recorded in       |      |
|       |     | U.S.A. from 1965 to 1977.                           | 4.0  |
|       |     |   | 40   |
| Table | (5) | : .% of different bacteria in umbilical swabs       |      |
|       |     | in first six days of life in both full term         |      |
|       |     | and premature newborns.                             | 44   |
| Table | (6) | : No. of premature infants colonized with           |      |
|       |     | different types of bacteria.                        |      |
|       |     | deficient types of bacteria.                        | 46   |
| Table | (7) | : Patients to whom silk sutures were applied        |      |
|       |     | to the umbilical stumps of their newborn            |      |
|       |     | infants.  | 65   |
| Table | (8) | : Patients to whom plastic clamps were applied      |      |
|       |     | to the umbilical stumps of their newborn            |      |
|       |     | infants.  |      |
|       |     |   | 67   |
| Table | (9) | : Comparison between clamp and silk suture groups   |      |
|       |     | as regard incidence of umbilical stump sepsis.      | 70   |

|       |      | <u>I</u>   | PAGE     |
|-------|------|--|----------|
| Table | (10) | : Comparison between male and female infants in clamp<br>group as regard incidence of umbilical stump<br>sepsis.                 | 70       |
| Table | (11) | : Comparison between diabetics and non diabetics in clamp group as regard incidence of umbilical stump sepsis.                   |          |
| Table | (12) | : Comparison between diabetics and non diabetics in silk suture group as regard incidence of umbilical stump sepsis.             | 72       |
| Table | (13) | : Comparison between patients with P.R.M. and other patients in clamp group as regard incidence of umbilical stump sepsis.       | 72       |
| Table | (14) | : Comparison between patients with P.R.M. and other patients in silk suture group as regard incidence of umbilical stump sepsis. | 73<br>73 |
| Table | (15) | : Comparison between male and female infants in silk suture group as regard incidence of umbilical stump sepsis.                 | 13       |
| Table | (16) | Comparison between age of mothers in clamp and silk suture groups.   | 74<br>74 |

INTRODUCTION AND AIM OF THE WORK

### INTRODUCTION

Umbilical stump sepsis is an important complication to the neonate. It is usually mild and present as a scanty purulant discharge. In more severe cases the surrounding skin may be red and oedematous. In rare instances the infection may lead to septiceamia, tetanus neonatorum, ascending hepatic infection and peritonitis which are considered a major cause of neonatal death due to infection.

## AIM OF THE WORK

To study the clinical, bacteriological and pathological changes of umbilical stump from different conditions of deliveries comparing silk suture with umbilical clamp.

REVIEW OF LITERATURE

## YMOTANA

Its diameter is I to 2.5 cm, with an average

covering is substituted, through which the three umbili-

except where it approaches the fetus an epithelial

Its exterior is dull white, moist and covered by amnion

fetal umbilicus to the fetal surface of the placenta.

The umbilical cord or funis, extends from the

cal vessels may be seen (Novak and Woodruff, 1979).

on the surface, or false knots, which are essentially than the cord itself, frequently create nodulations Folding and tortuosity of the vessels, which are larger length of 55 cm and a usual range of 30 to 100 cm.

varices.

broduce shortening of the vessel and thickening of acing spiral course so that, when they contract, they stimuli. The outermost muscle bundles pursue an interlcost which contracts readily in response to mechanical larly the arteries, are provided with a strong muscular and when fully developed the umbilical vessels, particuthe vein. When fixed in its normally distended state The two arteries are smaller in diameter than

the media, with folding of the interna and considerable narrowing of the lumen. This action may account for the periodic sharp constrictions of contour-the so-called valves of Hoboken which often characterize these vessels (Warwick and Williams, 1980).

The umbilical artery exhibits transverse intimal folds of Hoboken across part of its lumen. The mesoderm of the cord, which is of allantoic origin, fuses with that of the amnion. (Chacko and Reynods, 1954).

The umbilical vessels are rarely straight but usually show a twisted conformation which may exist as either a right-oraleft -handed cylinderical helix. The number of turns involved may be relatively few or, at the other extreme, may even exceed 300 turns. Their causation has been variously ascribed to unequal growth of the vessels, or the torsional forces imposed by fetal movements; their functional significance is obscure (Prichard and Macdonald, 1985).

#### **EMPRYOLOGY**

The yolk sac, and the umbilical vesicle into which it develops, are quite prominant at the beginning of pregnancy. At first the embryo is a flattened disc interposed between amnion and yolk sac. Since the dorsal surface grows faster than the ventral surface, in association with the elongation of the neural tube, the embryo bulges into the amniotic sac and the dorsal part of the yolk sac is incorporated into the body of the embryo to form the gut.

The allantois projects into the base of the body stalk from the caudal wall of the yolk sac or, later, from the anterior wall of the hindgut.

As pregnancy advances, the yolk sac becomes smaller and its pedicle becomes relatively longer. By about the middle of the third month, the expanding amnion obliterates the exocelom, fuses with the chorion laeve, and covers the bulging placental disc and the lateral surface of the body stalk, which is then called the umbilical cord, or funis. Remnants of the exocelom

in the anterior portion of the cord may contain loops of intestine, which continue to develop outside the embryo.

Although the loops are later withdrawn, the apex of the midgut loop remains its connection with an attenuated vitelline duct that terminates in a crumpled, highly vascular sac 3 to 5 cm in diameter lying on the surface of the placenta between amnion and chrorion or in the membranes just beyond the placental margin, where occasionally it may be identified at term.

The vessels in the cord at term normally are two arteries and one vein. The right umbilical vein usually disappears early during fetal development, leaving only the original left vein. Section of any portion of the cord frequently reveals, near the center, the small duct of the umbilical vesicle, lined by a single layer of flattened or cuboid epithelial cells. In sections just beyond the umbilicus, but never at the maternal end of the cord, another duct representing the allantoic remnant occasionally is found. The

intra-abdominal portion of the duct of the umbilical vesicle, which extends from umbilicus to intestine, usually atrophies and disappears, but occasionally it remains patent, forming Meckel's diverticulum (Pritchard and Macdonald, 1985).