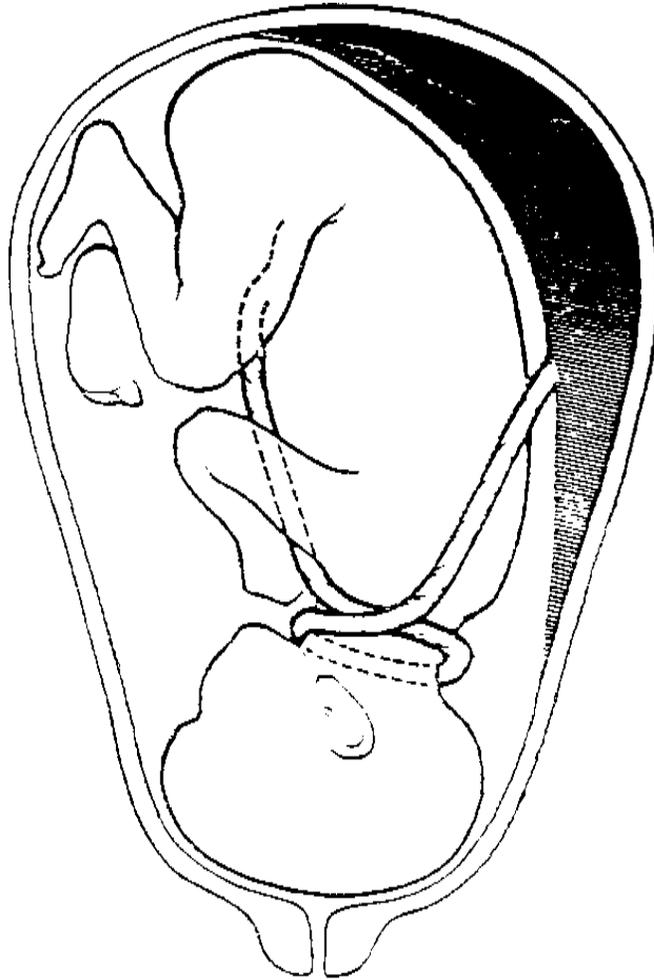


SILENT RISK

Issues about the Human Umbilical Cord



Jason H. Collins, M.D.
Charles L. Collins, M.D.
Candace C. Collins, M.D.

-- This Book is --

**Dedicated to the parents
who have experienced the loss
of a newborn secondary
to an umbilical cord accident.**

- Contents -

Acknowledgements

Preface

Introduction

Chapter 1 Origin of the Umbilical Cord

Chapter 2 The Umbilical Cord: Problem of Supply and Demand

Chapter 3 Fetal Behavior and Physiology

Chapter 4 What We Have Learned from Animal Study

Chapter 5 Management of the Umbilical Cord During Pregnancy

Chapter 6 The Future of Umbilical Cord Research

Recommended reading

– ACKNOWLEDGEMENTS –

We would like to thank the patients who provide us with the need to find a solution to the problem of umbilical cord accidents. Many parents courageously came forward and shared with us their experiences of umbilical cord related stillbirth. To discuss these events is difficult for them because to lose a normal fetus is so unique an experience.

Many scientists, researchers, physicians, midwives and nurses over the years have grappled with this issue. One who stands out in modern times is Jason C. Birnholz, M.D. who summarized the issue of the “supply line” (umbilical cord). In the context of an overall vision he states: “The practical goal of clinical obstetrics is to deliver an infant who will not only survive but develop without handicap from a prenatal or perinatal insult.”*

Oakbrook, Illinois

1990

This goal requires the application of tools called ultrasonography and fetal heart rate monitoring. Because of the efforts of Dr. Douglas Howry and Dr. John J. Wild, we can view the fetus in the uterus. Thanks to Dr. Edward H. Hon, fetal heart rate monitoring became established in obstetrical care. Many individuals have added to these great works and continue to do so today.

Now, many differing opinions exist about how effective these tools have been to reproductive care and “outcomes.” Let there be no mistake: to view the fetus and assess its physiology offers a chance to save a dying fetus where otherwise there would be no chance.

Many thanks go to Tulane University Medical Library whose holdings of journals, texts and special collections offer a repository of knowledge covering 200 years of obstetrical medicine. Margaret Verzwylt, Patsy Copeland and Cathleen Furlong provided much valuable time retrieving articles and hunting down rare interlibrary loans. Thank you to Mr. W. Postel for allowing the many unusual requests to hold his books at home for weeks.

Lastly, thank you to Jeanette Beauman, the Executive Secretary of the Pregnancy Institute, who typed everything, mailed everything and did everything to bring this book to reality. Thank you to Patricia Taylor who donated her editorial expertise and believed in the book.

*Ecologic Physiology of the Fetus, Ultrasonography of Supply Line Deprivation Syndromes, Radiology Clinics of North America, Vol 28, No 1, Jan 1990.

Preface

“Congenital abnormalities of the human umbilical cord and placenta may result in significant complications during labour. The ill-effects are predominantly upon the newborn - less common maternal morbidity and mortality may result.”

**Albert A. Earn, M.D., M.Sc.
Winnipeg, Canada
1951**

The issue of umbilical cord related fetal harm and fetal stillbirth is unaddressed in modern reproductive care. Although observations of umbilical cord related deaths have not necessarily proven causation, it is difficult not to ask “what is the relationship?” It is time to answer this question and to discern the full ramifications of umbilical cord related injury and death. The information discussed here is based on 8 years of research and a review of medical journals and texts. The authors have searched over 300 articles to provide a current panorama of this reproductive quirk which is not limited to humans. In addition, the Pregnancy Institute is dedicated to solving the problem of umbilical cord accidents. The Pregnancy Institute is a 501(c)-3 nonprofit medical research corporation co-founded by Jason H. Collins, M.D., an obstetrician interested in improving birth outcomes, Charles L. Collins, B.S.E., M.D., a pathologist interested in placental changes, and Candace C. Collins, M.D., a pediatric ophthalmologist interested in learning disabilities. By assembling this story it is our goal to persuade other researchers to turn their attention to the problem of solving umbilical cord accidents and anomalies. Hopefully the future will see a permanent solution. The mother, also, can play a role in solving the tragedy of umbilical cord accidents. While it is unknown how much time is needed for a fetus to die, it is believed that some fetuses die slowly. Fetal behavior is consistent and can have a repetitive (circadian) rhythm. As discussed later, awareness of fetal movements, sleep-wake cycles and tendencies may provide an initial warning of a compromised fetus. Verbalizing these changes to the obstetrician may alert everyone of the need for a closer look at the fetus with ultrasound and fetal monitoring. We hope that after reading this book you, the reader, will have a greater understanding of this tragedy. The expectant mother will hopefully understand her role in solving this tragedy.

“A survey of a number of British and American textbooks has yielded scant information on this subject [of umbilical cord complications].”

**Kan Pun Shui, B.S., M.B.
Nickolson J. Eastman, M.D.
Hong Kong, 1956**



(Figure 1) “Birthing Stones”, Oahu

Introduction

“It has always been a surprise to me that so little comment has been made on the large proportion of stillbirths which is associated with one or other of the various cord complications.”

**T. F. Corkill, M.D.
Wellington General Hospital
New Zealand
1961**

One has to wonder what thoughts prehistoric humans had when confronted with the still-birth of a baby entangled in its umbilical cord. Some insights from more recent times suggest the umbilical cord represented an omen, a sacred talisman, predictor of future fertility. In Europe, Australia, Africa, and Hawaii, the umbilical cord was dried and soaked in water for consumption to ensure future fertility. It was eaten, hung from tree branches, and stuffed in volcanic rock crevices at sites such as the “Birthing Stones” in Kukahiioko, Oahu (Figure 1). Chinese literature suggests the cord had medicinal properties.

European insights beginning with Galen (129 - 200 A.D.) suggested the umbilical cord served to nurture the fetus through arteries and veins. Leonardo da Vinci (1452 - 1519) observed that the cord was as long as the fetus at a given gestational age. Spiglius (1631) determined blood flow direction, and Harvey (1657) suggested that interruption of this blood flow could be the cause of fetal death if the cord was compressed.

Early descriptions of fetal loss from cord entanglement date as far back as 200 years ago. In 1750 the British obstetrician William Smellie (Figure 2) describes case #172 in Treatise on The Theory and Practice of Midwifery as a stillborn with four cords around the neck. (Figures 3 & 4). By the 1800s, many observations were recorded of distressed fetuses born with cord entanglement and cord abnormalities. A review of these early descriptions suggest clinical symptoms such as “pulling” sensations felt at the top of the uterus and excessive fetal movement followed by decreased fetal activity prior to fetal death.

Today, the field of obstetrics is confronted with the issue of umbilical cord complications - a timeless, almost prehistoric example of how imperfect reproductive evolution sometimes can be. Issues of birth-related blood loss, infection, and surgical intervention (C-section) have matured. Premature birth, congenital anomalies and toxemia still challenge the obstetrical community. Because umbilical cord accidents may represent a small number of fetal deaths, the motivation to



(Figure 2) *William Smellie*

investigate this reproductive tragedy may not be seen as urgent. However, out of 4 million births per year in the U.S., an estimated 4,000 umbilical cord related deaths occur. This is known as mortality.

What harm occurs to the live born fetus due to an umbilical cord complication is unknown. Obstetrical scientists call this harm morbidity. This morbidity is studied in terms of delivery “outcome,” meaning what harm is noticeable and how much. This harm often goes unnoticed for years. What harm does occur is rarely recorded.

Prenatal umbilical cord compression is currently suspected to provide such morbidity as neurologic damage. This damage may be as subtle as mild learning disabilities or as obvious as

SMELLIE'S TREATISE
ON THE
THEORY AND PRACTICE
OF
MIDWIFERY,
EDITED WITH AMENDMENTS
BY
ANDREW E. BUCHANAN,
M.D., F.R.C.S.,
OF THE GENERAL HOSPITAL, LONDON.
WITH NOTES AND THE HISTORY OF THE ART, BY
THE EDITOR.
LONDON:
THE NEW SYDNEY SOCIETY,
1881.

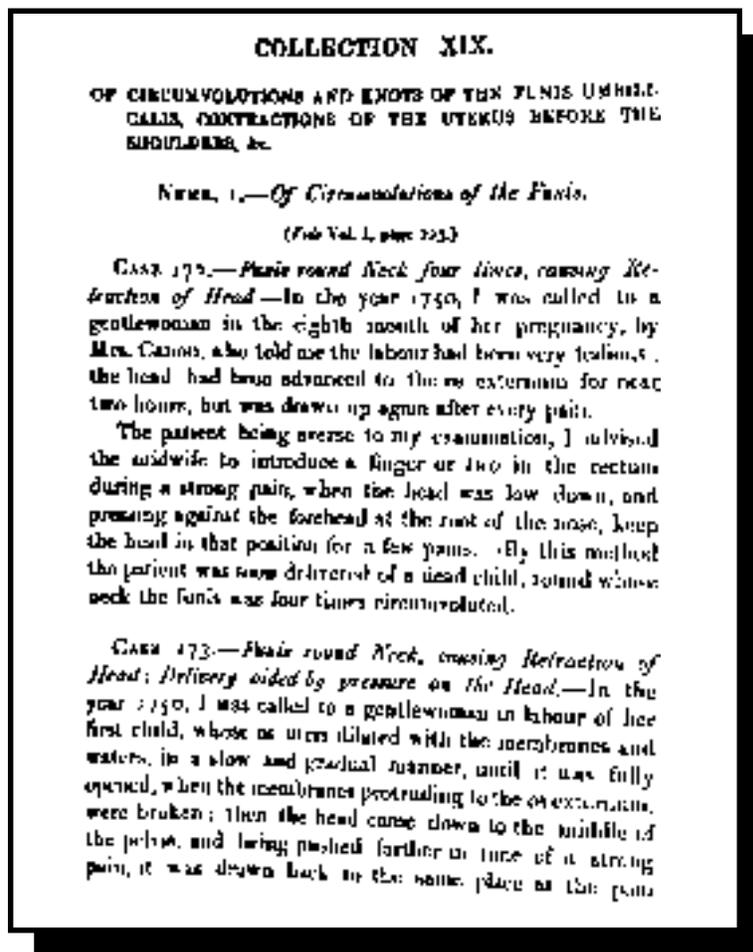
(Figure 3)

cerebral palsy. This is currently considered speculative by most, but not all, reproductive scientists.

The Perinatal Umbilical Cord Project (PUCP), an ongoing project at The Pregnancy Institute, seeks to understand the issue of umbilical cord complications, an event particularly tragic to the mother. If mothers are to be comforted, an explanation of how these events happen is important. The PUCP has established a scientific method (protocol) of observation and has prospectively inspected over 1,000 pregnancy cases. (Figure 5) ,

Method: All patients receive standard prenatal care starting with an exam at 8 to 10 weeks. This includes a vaginal ultrasound, a second ultrasound at 20 weeks, and a third ultrasound study at 28 weeks screens for umbilical cord problems. Also, at every visit the fetal heart rate is studied for 10 to 15 minutes and recorded. Patients identified with umbilical cord abnormalities (UCA) are watched bi-weekly. Repeat studies with ultrasound and fetal heart rate monitoring occur as needed.

As evidence and data accumulate, the authors hope that a solution can be created which will allow successful management of the normal pregnancy threatened by an umbilical cord complication.



(Figure 4)



FIG. 462.—Cranio Spina Bifida, seen by the Gross or Potts Method. Case in Chicago Lying-in Hospital Service.

(Figure 5)

Chapter One

Origin and Development of the Umbilical Cord

“The human blastocyst implants into uterine decidua... There is little realization that this supreme accomplishment is the final stage in several hundred million years of previous, step-by-step preparation of the blastocyst and the uterine mucosa for such a magnificent event.”

Richard Torpin, M.D.

(Student of the Human Placenta)

Medical College of Georgia

1931 - 1974

It has been estimated that 30% of births have some type of umbilical cord finding. This statistic implies a potential for fetal harm that may not be appreciated by scientific and public health authorities. Not knowing how many fetuses are harmed by their umbilical cords prevents research into the issue. If neurological harm can occur as the result of umbilical cord problems, then this mechanism of harm to the fetus needs to be investigated.

Every fetus should have the opportunity to begin life with all its God-given talents and abilities. Realistically, this may not be possible, but some physically normal newborns could benefit from a reduction in the risks of a cord mishap. It is estimated that learning disabilities may represent 15% of children today. What if one-third of these learning disabilities are due to some type of cord complication? The issue of cerebral palsy is important, but currently no solution and few insights exist as to its origin. Preventing the stillbirth of a normal infant would be an important step in identifying cord-related harm. What is the size of the problem, and what best describes each part of the problem of umbilical cord mishaps?

Disruption of the umbilical cord's supply line is a major source of harm to the developing fetus. It is estimated that every third to fourth delivery has an identifiable umbilical cord abnormality or anomaly. What is unknown is how these findings affect the fetus in degrees. The obvious effect is that stillbirth can result from the closing of the supply line.

The expectant mother can play a role in solving the tragedy of umbilical cord accidents.

While it is unknown how much time is needed for a fetus to die, it is believed that some fetuses die slowly. Fetal behavior is consistent and can have a repetitive (circadian) rhythm. As discussed later, awareness of fetal movements, sleep-wake cycles and tendencies may provide an initial warning of a compromised fetus. Verbalizing these changes to the obstetrician may alert everyone of the need for a closer look at the fetus with ultrasound and fetal monitoring.

To understand umbilical cord related complications, an understanding of fetal developmental physiology is imperative. The umbilical cord begins to form between four and six weeks, as the embryonic disc takes a cylindrical shape. Located at the lower third of the embryo, the proximal portion of the umbilical cord begins to form and develops a sac (herniation). The proximal portion houses the guts (intestines) until the tenth week of gestation. At this time the umbilical cord is short, usually shorter than the head-to-tail (crown-rump) length of the embryo and of proportionately large diameter. It is not able to tolerate rotation about itself or the formed embryo. In fact, as the umbilical cord elongates, the proximal cord encompassing the intestinal pouch cannot be disturbed. This initial stalk develops in the center of the implantation site, which is the reason the cord presents at the center of the afterbirth (placenta).

By ten weeks, the intestines leave the proximal cord and return to the stomach, the elongation of the cord begins, and the location of the umbilicus (bellie button) positions in the middle third of the embryo. The elongation of the umbilical vein and arteries coincides with the development of Wharton's jelly, an umbilical cord connective tissue. (Figure 6)

The responsibilities of the cord are numerous. For example, the cord manages its own growth, elongation, and expansion, accommodates increasing blood flow, and possibly assists the fetal heart. It also must regulate blood flow and its fluidity (thickness/thinness). In addition, the umbilical arteries and vein contain muscular coats that allow constriction of the vessels at birth or dilation of the vessels during growth. The umbilical cord also must produce its own chemistry to prepare for its role in birth and separation from the newborn umbilicus (a process which takes 7 to 10 days).

Located within the cord are the umbilical vein and arteries. The relationship of the umbili-



(Figure 6 - 10 week embryo)

cal vein to the umbilical arteries changes with development. These changes can result in cord abnormalities which will be discussed in the next chapter. Initially, two arteries send blood with waste products from the embryo to the afterbirth (placenta), and the one umbilical vein sends oxygen and nutrient-enriched blood to the embryo from the placenta. This circulation pattern must respond over time to the constantly changing fetal requirements and demands.

Rare developmental changes which can occur to the embryonic umbilical cord are persistence of the right “vitelline” vein, creating a four-vessel cord with two arteries and two veins. The reverse of this is obliteration of an artery and vein and the development of a two-vessel cord with one artery and one vein. There also exists a description of a “double cord” with separate vessel duplication.

Genetic problems are seen with two-vessel cords where fetuses with multiple malformations have defective organs which are not compatible with life. Maldevelopment of the genitourinary system such as bladder and kidney has been noticed. Although these relationships are known, obstetrics currently does not place any added concern on pregnancies with two-vessel cords. No remedy for these anomalies presently exists.

Umbilical cord vessels may multiply and branch under stressful conditions. For example, heavy smoking is associated with “multiple channels” in the umbilical cord. Hypoxia (the decrease of available oxygen) has been determined as the stimulus for opening of early “vestigial” vessels of the cord, once closed at 10 weeks. These findings of “re-channeled” embryonic vessels are also associated with fetal compromise and stillbirths. A trend was also noted in which first time pregnancies had more vascular branching than multiple birth mothers. Research indicates a 2% probability of this cord finding.

How the umbilical cord elongates and grows is unknown. As it grows, it changes the relationship between the vein and arteries. These changes may or may not predispose the growing fetus to blood flow disturbances or mechanical disturbances between the fetus and umbilical cord.

The umbilical cord is traditionally thought to “stretch” or “elongate” depending on the activity of the fetus. Active fetuses are believed to have longer cords on the whole than less active fetuses. Twins and triplets, because of restricted movement, have been shown to have cords on the average shorter than their single counterparts. Boys have longer cords than girls. Non-identical twins have varying cord lengths when compared to each other. Non-identical twin A can have a cord twice the length of twin B. Also twins A and B can have different cord architectures where one is straight and one is helical.

Rare instances exist in which no cord develops at all, the fetus being attached directly to the placenta at the umbilicus. Other reports in Chinese and French literature cite cords as long as 300 cm in length.

The umbilical cord appears to have organ-like properties. These properties are prone to disturbance under certain conditions which can affect the fetus. Just as a heart can fail pumping, or the liver and kidney can fail filtering the body's chemistry and waste products, the umbilical cord can fail in its role of being a "supply line."

Wharton's Jelly

Wharton's jelly is a specialized tissue serving many purposes for the developing fetus. Its specialized cells contain gelatin-like mucus that encase fibers. These properties give it an elastic and cushion effect, which can tolerate the vibration, bending, stretching and twisting of an active fetus. In addition, it holds the vessels together, may regulate blood flow, plays a role in providing nutrition to the fetus, stores chemistry for the onset of labor, and protects the supply line.

Umbilical cords without much Wharton's jelly are more prone to compression, and complete absence is usually associated with fetal death. If an umbilical cord is twisted or knotted, it is more likely to tighten where there is less resistance, such as an area low in Wharton's jelly.

It is believed that males have more Wharton's jelly content than do females and that good nutrition increases the amount. Wharton's jelly tends to reduce with gestational age and can disappear when pregnancies go beyond 40 weeks. Because these cases tend to have fetal heart rate changes, the level of Wharton's jelly is a consideration when obstetricians plan the deliveries of pregnancies low on amniotic fluid.

Cord Length

Cord length can be associated with neurologic abnormalities and fetal positioning. To understand this correlation, it is important to understand the physiology of the human umbilical cord. Cord length has been frequently measured. One of the largest studies was completed by Pathologist Dr. Richard Naeye. In his book *Disorders of the Placenta, Fetus, and Neonate*, Dr. Naeye averages the lengths of different umbilical cords at progressively older gestational ages. The cord is believed to elongate until as late as 36 weeks although rapid change occurs until 28 weeks, then slows. The final length of the umbilical cord averages about 61 cm, or 24 inches, according to Percy Malpas, M.D., a British obstetrician who studied cord length in the 1960s. The first preg-

nancy tends to generate a shorter cord than subsequent pregnancies. Although no published report of a genetic relationship exists, there may be one.

So why 61 cm? Umbilical cords of whales, porpoises, goats and other mammals are relatively shorter than the human cord. Walker and Rye of Cambridge surmised in the British Medical Journal in 1960 that prehistoric humans evolved length for protection. Nature's purpose was to allow the mother to pick up the newborn without disturbing the placenta. The event of breast feeding would then separate the placenta - an event which could attract predators. Having the fetus in tow would allow escape for mother and child.

Today, cord length correlates to several "outcomes." Cords too short and cords too long predispose the fetus to intrauterine dangers. A short cord has a length of less than 32 cm. This length was determined in 1910 by a famed Chicago obstetrician, Dr. Joseph B. DeLee. Dr. DeLee believes 32 cm to be the minimal length necessary for a term fetus to deliver. The concept changes, however, when cord insertion site and cord entanglement are considered. This idea is called a relatively short cord. Very short cords less than 20 cm are associated with genetic malformations. When cord lengths were evaluated for IQ, short cords showed a higher incidence of neurologic abnormalities.

Cord length may also influence fetal position. Torgrim Sørnes, M.D., a Norwegian researcher, observed this. His work suggests that Breech-positioned fetuses have shorter cords due to less activity. This insight suggests that if the fetus persists at remaining Breech, a cord etiology should be considered, and the obstetrician should watch for fetal difficulties during labor.

Umbilical cord circumference and diameter are also important measurements. On average, normal umbilical cords are 3.7 cm in circumference with a range of 3 - 5 cm. The diameter range of 1.0 cm to 3.0 cm can suggest an abnormal cord with edema, tumor, or hernia. Dimensions greater than 6 cm circumference should prompt an examination of the cord and fetus. Are shorter cords thicker than longer cords? Although rarely published, it appears that this may be the case. Before cutting any thick cord, it should be checked to ensure that the fetal intestine is not present within the cord.

Growth and development of the umbilical cord are dependent upon many factors. Disturbance of these events can lead to fetal compromise or result in fetal compromise. These effects will be described in the next chapter.

Umbilical Cord Design

How the umbilical cord is built has long been of interest to anatomists. A look at all mammals shows a variety of design adaptations. In humans, it has been determined that there are several designs. What these differences mean to the fetus is unknown. Attempts by several noted scientists to understand how the umbilical cord works have taught us that the cord is more like an organ rather than a rigid conduit (pipeline).

Not all cords are alike. Just as there are different kinds of hair (curly/straight, thick/thin), there are different kinds of cords. Most cords (99%) have three vessels, although some (1%) have only two, and even less have four.

The relationship between the normal vein and two arteries is usually parallel (Figure 7). This parallel configuration can vary, however, and may imply effects which can alter blood supply to the fetus. Variances include arteries that are together, or separated with each artery lateral to the vein (Figure 8). Another variance is arteries that wind around the vein while the vein remains central in the cord. This is sometimes referred to as spiralled arteries, but helical is the preferred term (Figure 9). The vein can also parallel the arteries in a helical configuration, and the vein can wind around the arteries.

Several researchers have concentrated on these differences and suggest that umbilical cords of absolutely straight designs may be more prone to disruptions of blood flow. If these observations are verified, it may be important to know the cord design prior to delivery.

The location of umbilical cord attachment to the fetus and placenta is also important. Placental attachments can be in the center, off center, on the edge, or in the membranes. Membranous insertions of the umbilical cord are called Velamentous Insertions. These placental-cord designs have flaws which can lead to cord tears. Currently, little research has been done to □ develop prenatal diagnostic criteria.

Umbilical attachment of the cord can vary and predispose the infant to hernias at the umbilicus and “constriction” of the cord. Although these are uncommon findings, future research will allow a more accurate evaluation of the umbilicus. Amniotic bands can interfere with both ends of the umbilical cord. For example, the amniotic membrane can leave remnants in the form of fibrous bands which can stiffen and occlude the blood circulation through the cord. These events are reproductive mishaps that have no current remedy. In order to begin the process of creating solu-



Figure 7
A normal vessel pattern consisting of two umbilical arteries and one vein.

Figure 8
A different parallel vessel pattern with a straight vein and parallel arteries.



Figure 9
A different view of an artery relationship of a straight vein with helical arteries wrapped around the vein.

tions to umbilical cord related □ complications, understanding cord function and design must be thorough.

Umbilical Cord Abnormalities and Anomalies

Throughout human history, stillbirths have been associated with umbilical cord findings. These findings vary, and some are more common than others.

Scientifically, umbilical cord changes and effects are described several ways. To start, the umbilical cord can develop design flaws which can lead to fetal harm. These flaws are called Umbilical Cord Abnormalities:

Table 1

Short Cords - less than 35 cm

Long Cords - more than 70 cm

Two Vessel Cords - one artery / one vein

Four Vessel Cords - two arteries / two veins

Velamentous Insertions - inserted on the membranes

Marginal Insertions - inserted on the Placental edge

Constriction of the Umbilicus - lack of Wharton's jelly at the fetal insertion

Straight Cords - parallel arteries and vein with no Wharton's jelly

Wharton's jelly cysts - Mucinous
- Myxoid
- Edema

Growths of the umbilical cord

- Umbilical Artery Angioma/aneurysm**
- Umbilical Vein varixes / False knots**
- Hematoma/Teratoma/Thrombosis/rupture.**

These abnormal umbilical cords are predisposed to rupture, mechanical failure, entanglement, disruption of labor, uterine malfunction, and premature labor. The ultimate effect is disturbance of the life line and derangement of blood flow to the fetus. The difficulty lies in the fact that these abnormalities are silent and invisible.

Short Umbilical Cords (less than 35 cm) are predisposed to rupture and prevention of fetal descent during labor. Very short cords, less than 25 cm, are associated with genetic malformations. Short umbilical cords need to be considered relative to their attachments to the placenta. The further the attachment is from the cervix, the less likely the fetus can be

born vaginally, requiring a C-section.

In addition, fetal heart rate changes will be more likely to occur during monitoring, creating concern for all involved in the labor process. Very short umbilical cords, less than 25 cm, have been associated with neurologic disorders, IQs less than 80, and cerebral palsy. There is an increase in stillbirth risk with short and relatively short cords. This risk may be as much as six times more likely, especially when other factors like toxemia are involved. Short cords and cigarette smoking tend to result in small babies, called IUGR (Intrauterine Growth Retarded). It is difficult to unravel the relationships mentioned above since some fetuses may incur neurologic damage which predisposes them to decreased activity and leads to decreased cord length.

Long Umbilical Cords (longer than 70 cm) are associated with a number of circumstances which can impact fetal life. Leonardo da Vinci studied cord length and believed it was a proportional/natural relationship of 1 to 1 (cord length = fetal age in weeks). Although this is not precisely correct, da Vinci was correct in that it is proportional. Biological and physical principles which dictate the shape of a star fish, tree leaf, or nautilus shell determine the positive or negative relationship between the fetus and its umbilical cord (and probably placenta).

Fetal activity is believed to determine umbilical cord growth. This mechanical stimulus may be a direct or indirect factor. How does the umbilical cord grow and elongate? Biochemical and cellular mechanisms must be at work. All of these molecular-genetic pieces are potentially at risk for failure by inside or outside disturbances. Growth factors have been identified in the umbilical cord. In addition, studies in twins suggest a genetic control or modulation of length. Length can also be influenced by amniotic fluid volume and anything that constricts fetal movement. Umbilical cords are also innervated to a degree near the umbilicus. The role this plays or whether there is an influence on cord development is currently unknown. Of all those multiple variables influencing cord length, the most important variable needs to be determined. It is unknown whether individual cell enlargement or cell division and multiplication cause cord growth. Many different cells such as muscle cells, endothelial cells, fibroblasts, connective cells and amniotic cells all must do the same thing. Insight into this aspect of fetal development may help understand anomalies of the cord.

Microscopic comparison of long and short cords may reveal differences of structure. Thickness or thinness of vessel walls, composition of Wharton's jelly, and artery-vein interrelationships may be important findings which explain long cord susceptibility to various events.

Two Vessel Cords occur in about 1% of births. The connection to fetal harm or well-being is unclear. These umbilical cords have one artery and one vein. The dominant artery origination

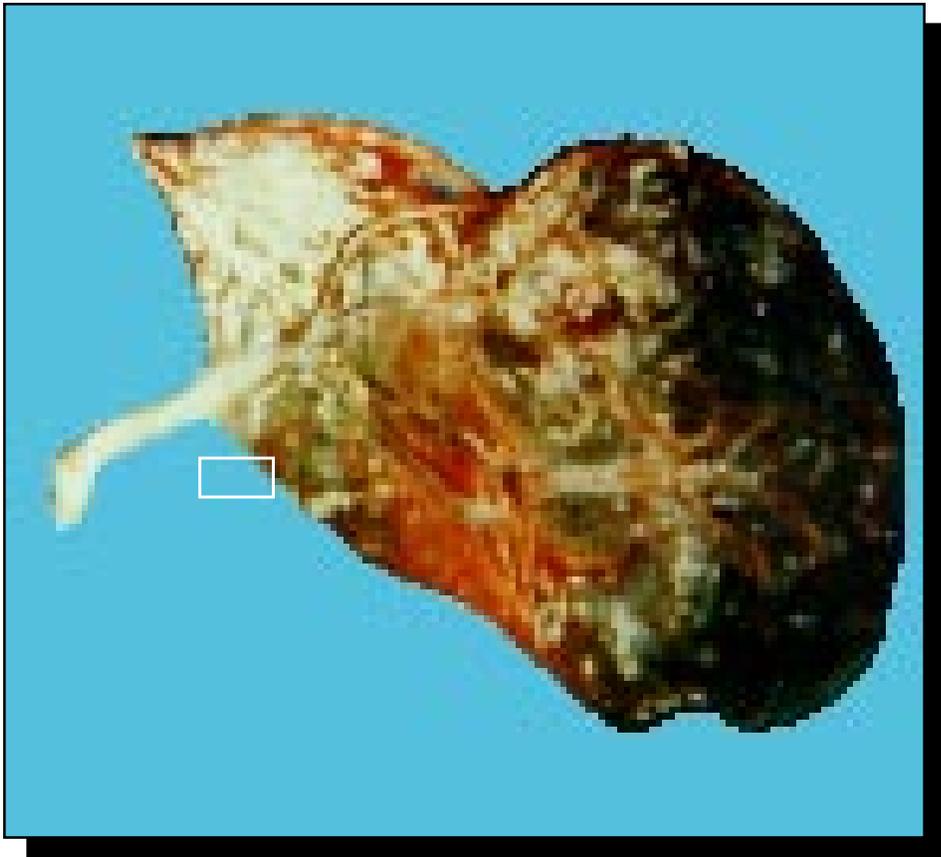
(left or right inside the fetus) determines whether or not congenital malformations may be present. It is accepted that these cords may predispose the fetus to stillbirth compared to a normal three vessel cord. The risk of stillbirth can be six times greater than normal especially when other factors such as toxemia exist. Whether or not other variables are involved remains to be determined. Development of a single umbilical artery cord may be associated with maternal smoking, drug exposure, placental abnormalities and maternal diabetes. Whether or not all infants with a two vessel cord are predisposed to some difficulty remains to be seen. The mechanism of how one artery is obliterated versus undeveloped may be important to understanding this issue.

Four vessel umbilical cords are rare and are mentioned to emphasize the vulnerability of cord vessels to malformation. Not all umbilical cords are alike, and non-identical twins can have non-identical cords. Proper development of the embryo and its supply line are important steps toward a healthy fetus and newborn. Maldevelopment of the supply line from the start can predispose the fetus to harm.

Another important step in umbilical cord development is the connection of the fetus and placenta to each other. The fetal connection is specialized and has a specific architecture. This design needs to function as a secure tether for the fetus, as a disrupter for umbilical separation (in mammals the cord tears free or is chewed free), perhaps as a sensor for blood flow into the fetus, and must merge with the skin.

Researchers have identified nerve endings near the umbilical insertion of the cord in the Wharton's jelly. These "end nests" may play a role in communicating with fetal "valves" called shunts relative to blood volume wave properties entering the fetal circulation through the umbilical vein at the level of the liver and heart. When the umbilical end is malformed, constriction or coarctation occurs, stopping blood flow. How this happens is unknown. At the other end of the supply line, the fetal arteries enter the placenta with a membranous support tether and distribute in a branching manner. When the placenta develops it sometimes "migrates" and "dissolves" from its original site. This sometimes can result in what appears to be a relocation of the placenta. The placenta tissue dissolves, leaving a membrane (the amnion) remaining which can then be the connection (insertion) site of the umbilical cord. This results in the umbilical cord placental end looking like it is connected to the edge of the placenta (called a marginal or Battledore insertion) and a membranous insertion called a (Velamentous) insertion. (Figure 10)

Another variation is called a furcate cord insertion in which the cord does not connect to the placenta but its branching elements do; however, no membranous insertion exists. These malfor-



***(Figure 10) Velamentous Cord Insertion
(Arrow shows vessels in the membranes instead of in the placenta)***

mations account for another 0.5% to 1% of all births and are observed increased in premature labor, premature birth, fetal stillbirth, and neurologic harm.

The risk of cord vessel rupture is increased with an abnormal cord insertion. The difficulty of managing an incident such as cord rupture is great. What makes the mystery even more complicated is the location of the cord insertion in the uterus. If the membranous insertion is over the cervical opening, the risk of tearing and fetal blood loss is great. If a marginal insertion is against the sacrum (lower backbone), the risk of compression and fetal circulation disruption is great as the fetus descends into the pelvis. This relationship of vessel location to fetal location has caused sudden fetal distress and the need to activate a surgical team for an emergency C-section. It is unknown how often this happens during day-to-day obstetrical care as the attention is on the fetus, not the placenta, its location, or its cord insertion architecture.

Umbilical cords may have eight different types of design. The extremes are very helical cords (95%) and completely straight cords (5%). The association between umbilical vein and arteries can vary where veins wind around arteries, veins and arteries are parallel, and arteries wind around veins. The veins can be parallel with the arteries as well (10%). Very helical designs (spiralled, coiled, and curled) may predispose the fetus to certain blood flow changes, and very straight designs may be susceptible to compression. It is unknown what the fetal effects are but some evidence points to supply line vulnerability when the design is faulty. Add to this other variables such as placental location and umbilical cord insertion site, and the combination becomes a significant factor in determining the well-being of the fetus. Knowing these details may provide important insights into the development of fetal harm.

Wharton's jelly, although apparently inert looking, may be an important chemical factory for the fetus. Additionally, its components and cellular make-up can predispose the fetus to the formation of tumors, cysts, and edema. Edema is not an infrequent finding at delivery of a newborn (10%). It is usually limited to small sections of the umbilical cord and associated with trauma due to fetal behavior. Extensive involvement of the cord is associated with complications of pregnancy such as toxemia and infections. When cause is due to fetal circulatory disturbances, fetal heart failure may predispose the cord to edema which is associated with an increased risk of stillbirth. Tumors can develop in Wharton's jelly. Although rare, "teratomas" have been reported. Teratomas grow to large sizes and can disrupt vessels and blood flow.

Embryonic features of the umbilical cord can produce six types of remnants, some of which look like "hemangiomas," blood vessels which together look like small varicose veins in a bundle.



Figure 11
A normal vessel
pattern consisting of
two umbilical arteries
and one vein. Seen in
cross section with it's
Wharton's Jelly.

Figure 12
An alternate vessel
pattern, in cross section
with the vein around the
arteries and no
Wharton's Jelly.



Figure 13
An abnormal vessel
pattern
consisting of one artery
and
one vein.

Other structures such as “Viteline duct remnants” and “Urachal duct remnants” can be seen. Those changes are very infrequent but should be considered if an enlargement or localized “mass” is seen in the cord. Hematomas (bleeds into the substance of the cord) can occur and mimic these rare tumors. The possible association with fetal defects must always be considered. As the fetus ages, it is believed that Wharton’s jelly recedes. This becomes an important issue when deciding to deliver a “post date infant” where the due date has been passed without delivery. Loss of Wharton’s jelly may put the fetus at risk of cord compression and therefore fetal harm. (Figures 11, 12, 13)

Growths and Swellings of Umbilical Cord Vessels

Like any vessel in the human adult body, umbilical cord vessels can develop sacs, protrusions, bulges, and varicosities. For example, cord vessels can protrude and thrombose like varicose veins or hemorrhoidal veins. In addition, the umbilical vein sometimes bunches up on itself, creating the appearance of a “false knot” or of multiple varicosities; these spaces can act like quiet pools of blood that can clot and predispose the fetus to a thrombo-embolism. The clot can break free and enter the fetal circulation or can obstruct cord blood flow. The umbilical arteries can develop similar pockets called “aneurysms.” These bulges in the arterial vessel wall can rupture and lead to fetal hemorrhages in the uterus. These alterations of structure can predispose the cord to rupture as well.

Human umbilical arteries consist of two layers of muscle fibers, the outer layer being 3/4 of the wall thickness and an inner layer. The design of the muscle cells is parallel where the inner layer runs with the vessel lengthwise, and the outer layer surrounds the vessel like a spiral staircase. A thin layer of cells line the vessel opening, and an outer layer is formed by connective tissue and Wharton’s jelly. This architecture allows constriction and shortening of the vessel. Defects in this structure can occur which may predispose the umbilical arteries to failure.

A type of architectural defect is called “umbilical cord vessel segmental thinning.” In this malformation (1%), umbilical vessel walls are missing a layer of muscle, therefore weakening the vessel. Related observations include fetal anomalies and perinatal problems. These fetuses are predisposed to stillbirth, meconium, and fetal heart rate decelerations.

All in all, when a combination of defects results, the risk of umbilical cord failure begins to become important. It is unknown how many placentas and umbilical cords contain a variety of architectural anomalies or abnormalities that lead to miscarriage. It is unknown how much fetal harm may be the result of faulty placentation and cord alterations such as straight cord segments

resulting from the molding of Wharton's jelly due to long-term compression of an entangled cord. Future research into these issues will be both exciting and fruitful. The integration of the anatomy (structure), biochemistry (substance), and physiology (function) of the umbilical cord will allow the emergence of a new awareness of three structures to manage in pregnancy: the placenta, umbilical cord, and fetal unit.

How the Human Umbilical Cord Works (Physiology)

What is remarkable about the umbilical cord is that it is a blood vessel without branches. This is unique compared to the large blood vessels of the adult body, the aorta and vena cava. Its properties, therefore, are different in some respects and alike in others. The umbilical cord has two-way traffic: the arteries carry blood pumped by the heart away from the fetus, and this circulation surrounds the vein normally; the umbilical vein returns blood to the fetus from the placenta rejuvenated with oxygen and nutrients and devoid of waste products.

How this happens is still surrounded by mystery. The fetal heart cannot expand or work harder because it is surrounded by a fluid-filled lung, like pushing against a water bed. Therefore, as the fetus steadily grows exponentially and three-dimensionally, how does it accommodate the increased blood volume it needs over time? As the fetus grows, the cord elongates and grows in diameter. The fetus has to work against a larger column of fluid and tissue resistance at the placental end. It has been estimated that by 31 weeks, the umbilical cord must carry 70 quarts of blood per day, moving at 4 miles an hour.

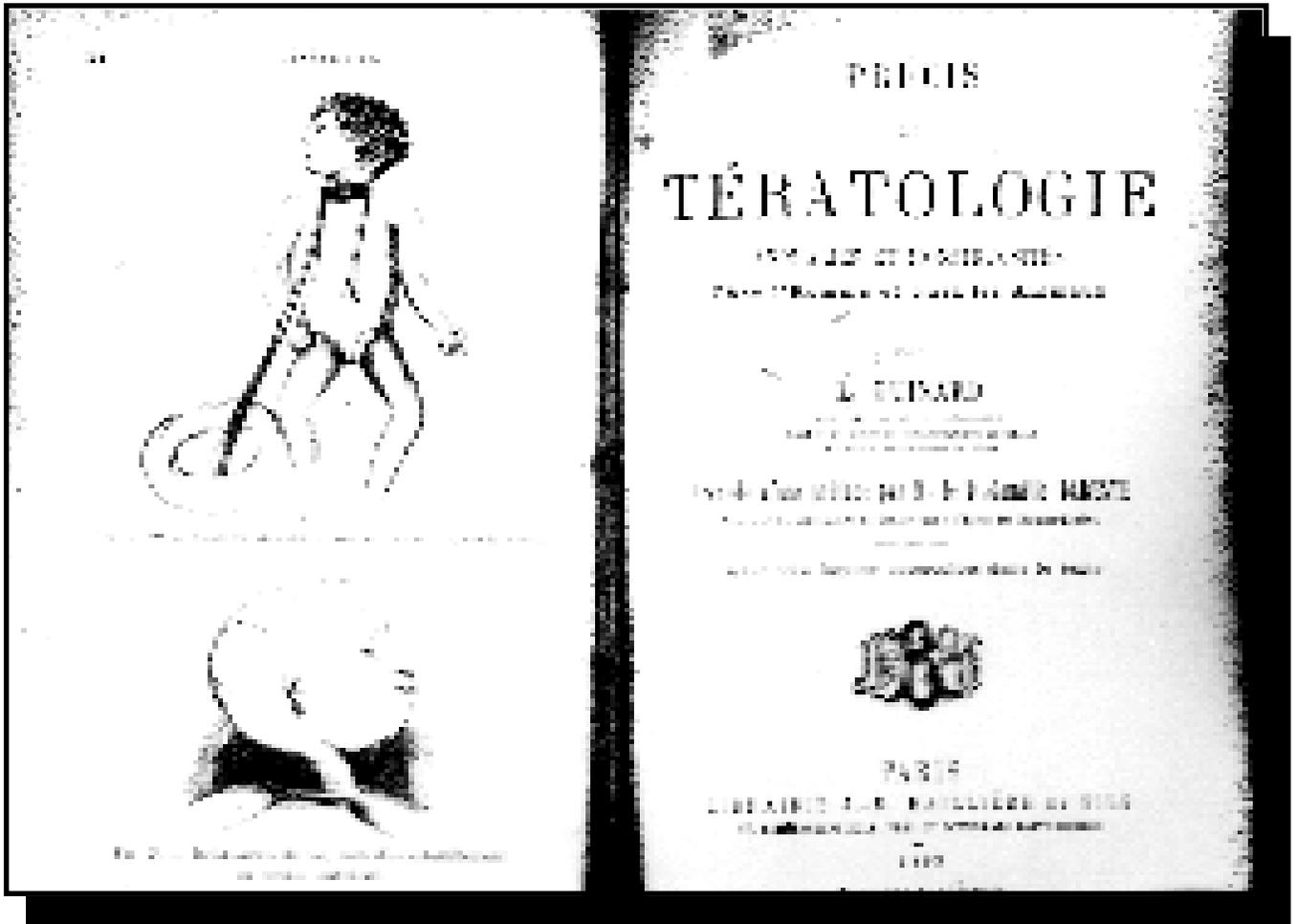
This remarkable organ also must participate in fetal growth milestones; additionally, it may act as an assist pump to the fetal heart. This assist pump may be designed to help the fetus over difficult growth proportions which may exist at 20 weeks, 24 weeks, 28 weeks, and 32 weeks - times that are known for premature labor to appear. The extra stress on the fetus may require that the cord be designed correctly so that it can have properties of an assist mechanism or pump. This theory, proposed in the 1950s, requires that the arteries surround the vein in the proper architecture. If this is so, then future research into this issue may explain fetal effects secondary to cord design. To date, no assist pump property has been detected in the umbilical cord.

How blood flow is regulated in addition to being carried by the umbilical cord is unclear. Cord length does not significantly affect blood flow dynamics; however, blood flow must meet some resistance for the circulation to work. As a result, the umbilical arteries are surrounded with four layers of smooth muscle to maintain a certain amount of muscular tone. The umbilical vein is

not as muscled. The system operates fully dilated, but stimuli from chemistry or hormones can affect the system and cause constriction. This must happen at birth to reduce blood loss. In larger mammals, the cord must constrict from the placenta to the fetus for the fetus to avoid anemia. In the human, similar mechanisms may be available chemically. Regulation of blood flow, vessel constriction at birth, and blood loss prevention may be the roles of these vessel-active substances. Some of these substances originate in the placenta.

Researchers using ultrasonography recently have been able to measure umbilical blood flow with color Doppler imaging. This technique allows visualization of the blood vessels based on the movement of the blood itself. These studies also suggest that the umbilical vein, arteries, and placenta act as an assist pump of sorts to the fetal heart. Measurement of blood flow allows the obstetrician to determine whether enough blood volume is circulating in the placenta to provide nutrition and oxygen to the fetus. Under certain conditions this blood flow can be reduced and circulation in the placenta altered to create a growth-affected fetus, Intrauterine Growth Retardation (IUGR). In essence, it is a way of determining the fetus' blood pressure.

These findings become important because, in addition to the potential for fetal harm or stillbirth, important lifetime tendencies are emerging. The fetus seems to have the ability to set its vital signs for its adult life. If stressed, the IUGR fetus sets blood pressure and heart function, which can predispose the fetus to adult heart attack. These mechanisms are just beginning to be understood, and the umbilical cord may be an important part of the mystery.



A drawing of a term stillbirth with a straight umbilical cord design with an abnormal placenta, a marginal umbilical cord insertion, and a double nuchal cord. (A case report from Paris, France, 1893)

Chapter Two

The Umbilical Cord: The Problems of Supply and Demand

For the layman and more so to the obstetrician, [the umbilical cord] presents a potential hazard during delivery—It is highly desirable that umbilical cords should be examined at delivery.”

Bholanath B. Nadkarni
Professor of Pathology
University of Ottawa/Ontario
Canada, 1969

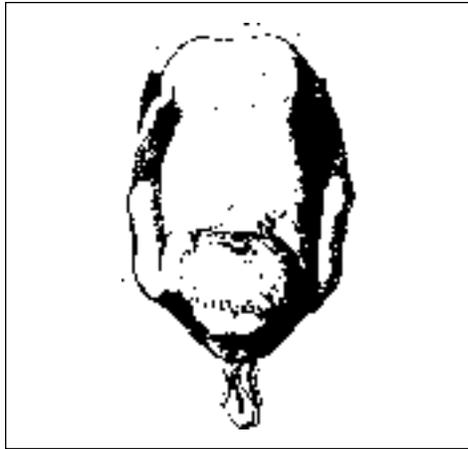
“Many pathologic features of the umbilical cord affect fetal well-being adversely.”

Kurt Benirschke, M.D.
Professor Emeritus
Department of Pathology
University Medical Center
San Diego, California, 1994

Disruption of the umbilical cord (the fetal supply line) may be a source of harm to the developing fetus. An estimated 30% of pregnancies carried to term have some type of umbilical-placental abnormality. This means that every third to fourth delivery has an identifiable umbilical cord abnormality or anomaly. What is unknown is how these findings affect the fetus and to what degree. This chapter discusses the types of umbilical cords and their potential inherent complications.

The obvious unwanted effect of supply line disruption is stillbirth. The most dramatic example of this obstruction is an emergency called “prolapsed umbilical cord.” Prolapse of the cord occurs when a fetus is not properly positioned in the pelvis. Membranes rupture and wash the cord out of the cervix in front of the fetal buttock or head. When the fetal body engages the cervix, the cord is compressed, and blood flow to the fetus ceases. If the patient has the good fortune of being near an operating room, the fetus has 10 minutes before harm begins. If a C-section can be performed quickly enough, the infant usually survives without harm. (Figure 14)

Many variations of this event exist in which factors such as time, degree of compression, and age of the fetus play a role. Yet the common thread among these variations is immediate danger to the fetus. Compression of the umbilical cord obstructs blood flow to and from the fetus. This total obstruction denies the fetus oxygen and blood flow pressure, and it releases stress factors from endocrine organs which contribute to fetal harm.



(Figure 14) A developing prolapse cord.

Umbilical cord compression clearly plays a role in disturbing the well-being of the fetus. The effects of incomplete cord compression are unknown. These impacts are altered by different effects at different gestational ages under various conditions at varying intervals. Current obstetrics continues to debate these issues. Although stillbirth clearly can be caused by a variety of mechanisms creating cord compression, prolapse of the umbilical cord is the only “witnessed” event that is acknowledged and accepted as a fatal cause-effect complication. The chance of having a pro-

lapsed cord at birth is 1 out of 239 cases to 1 out of 865 cases. The reported chance of fetal loss from a prolapsed cord is 8.6% to 49% of these prolapse occurrences. Similar mechanisms can occur inside the uterus with the same results. One such occurrence is that it can take place with “monoamniotic twins.” These twins have no barrier between them and can entangle their cords. Stillbirths often occur in one twin or both because of knots and encirclement. It is accepted that these mechanisms do cause stillbirth. On the basis of this knowledge, examination and anticipation of other cord complications is not difficult. (Table 1)

Obstetrical thought now accepts that “monoamniotic” twins can interfere with each other’s umbilical cord. Pregnancies of “monoamniotic” twins showing no other risk factors or complications are monitored carefully for this supply line disruption. Published case reports have been described of entangled twins with multiple knots. Intensive surveillance was needed for these monoamniotic twins. These reports have described early intervention when fetal heart rate changes suggested the need for delivery as early as 31 weeks. If this concept is recognized for monoamniotic twins, why is it not recognized for single fetuses with cord anomalies or abnormalities?

Table 1
Worldwide frequency (incidence) of umbilical cord events and stillbirths (mortality)
To date a world literature review suggests the following:

UCA	Incidence	Mortality
Single umbilical artery	.2% - 3.6%	7%
Non-coiled cords	4.3%	Unknown
True Knots	1%	6%
Nuchal Cords	14% - 30%	Unknown
Body Loops	1%	10%
Short Cords	Unknown	Unknown
Velamentous Cords	.54% - 2,17%	30%
Torsioned cords	6%	20%

Regardless of mechanisms such as prolapsed cord or twins, the “pathophysiology” is the same: supply line deprivation, restriction of blood flow, and reduction of oxygen and nutrients to the fetus to the degree that injury or death can occur. What does such a mechanism look like? Again, 30% of fetuses delivered have some finding. What are the varied types of cord abnormalities and accidents that can affect the fetus? Some observations from history give us clues. (Table 2)

Leonardo da Vinci was not only an artist, but a sculptor and engineer. He recognized in the human form the same principles of proportion as seen in nature. The Greeks had defined many proportions and believed them keys to understanding life.

The fetus is proportioned to itself. The early fetus is seen in thirds and the growing fetus in fourths. The term fetus can be seen in fifths and it can develop entanglement once the umbilical cord is proportionally long. This seems to suggest the point at which the cord begins to pose a danger. This point begins with the cord being $4/5$ as long as the fetal head-to-toe length. At this length the neck can be encircled 360 degrees. This can often happen after 10 to 12 weeks gestational age. A cord of lesser proportional length can't completely encircle the neck.

Cord length plays a role in how a fetus develops, how labor is tolerated, and how delivery occurs. What are the different cord length effects that have been reported in the medical literature? **Absent Umbilical Cords:** If the umbilical cord does not develop, the fetus can develop but can be malformed. The fetus is directly attached to the placenta at the abdomen and usually develops defects. Fetuses without cords have been born by C-section. Without an umbilical cord, life is usually not possible. Yet this concept was not always apparent. The roles played by the placenta, cord and fetal heart were not realized until the late 1700s.

Short Umbilical Cords: A cord can be short (20 cm) but can allow delivery if it is attached to the placenta near the cervix. Yet as previously described, short umbilical cords can affect the fetus. Restriction of movement may occur for genetic or other reasons. Fetal movements may predispose the cord to compression, constriction and, during labor, failure to progress through the birth canal. Short cords can also be responsible for fetal distress during labor, separation of the placenta, and rupture of the umbilical cord prenatally or during labor. In addition, short cords can predispose the umbilical vein and arteries to tears which can lead to hematomas (vessel rupture) of the cord. Inversion of the uterus (inside out and a medical emergency) can occur from traction on a short cord. Dr. Smellie describes a case of this type. (Figure 15)

A relatively short cord is a cord that is entangled with the fetus. Although the length is normal, it is caught over the shoulder or around the arm or neck and uses up “slack,” becoming

CASES 174, 175.—*Delivery retarded by Shortness of the Fetus.*—I have in this matter assisted in a few cases where delivery was retarded by the shortness of the fetus; particularly in the year 1744, when the patient was delivered by the forceps, and in the year 1750, when the woman was delivered by the labour-pains, assisted in the manner described above; in this last case the fetus was not above two hands-breadth long, though very thick.

Mauriceau, in p. 336, and Obs. 456, relates an instance of his having delivered a woman of her first child, whose navel-string was extremely short, and as thick as its arm. The child had been dead several days before delivery.

It may be proper to observe, that when labour is retarded by the shortness or circumvolution of the fetus, the retraction or drawing back of the head does not begin to be perceived until it is low in the pelvis, whereas it is sooner observable when owing to the contraction of the uterus before the shoulders.

The head is also low down before it can be retarded by one of the shoulders resting above the os pubis or sacrum, instead of being towards the sides at the brim of the pelvis.

(Figure 15) Dr. Smellie's case 174 of a short cord.

Table 2

mechanically unable to allow vaginal delivery. Disruption, compression, and rupture of the cord can occur in a cord that is relatively short. Prenatally addressing a condition of this type will be discussed in Chapter 5.

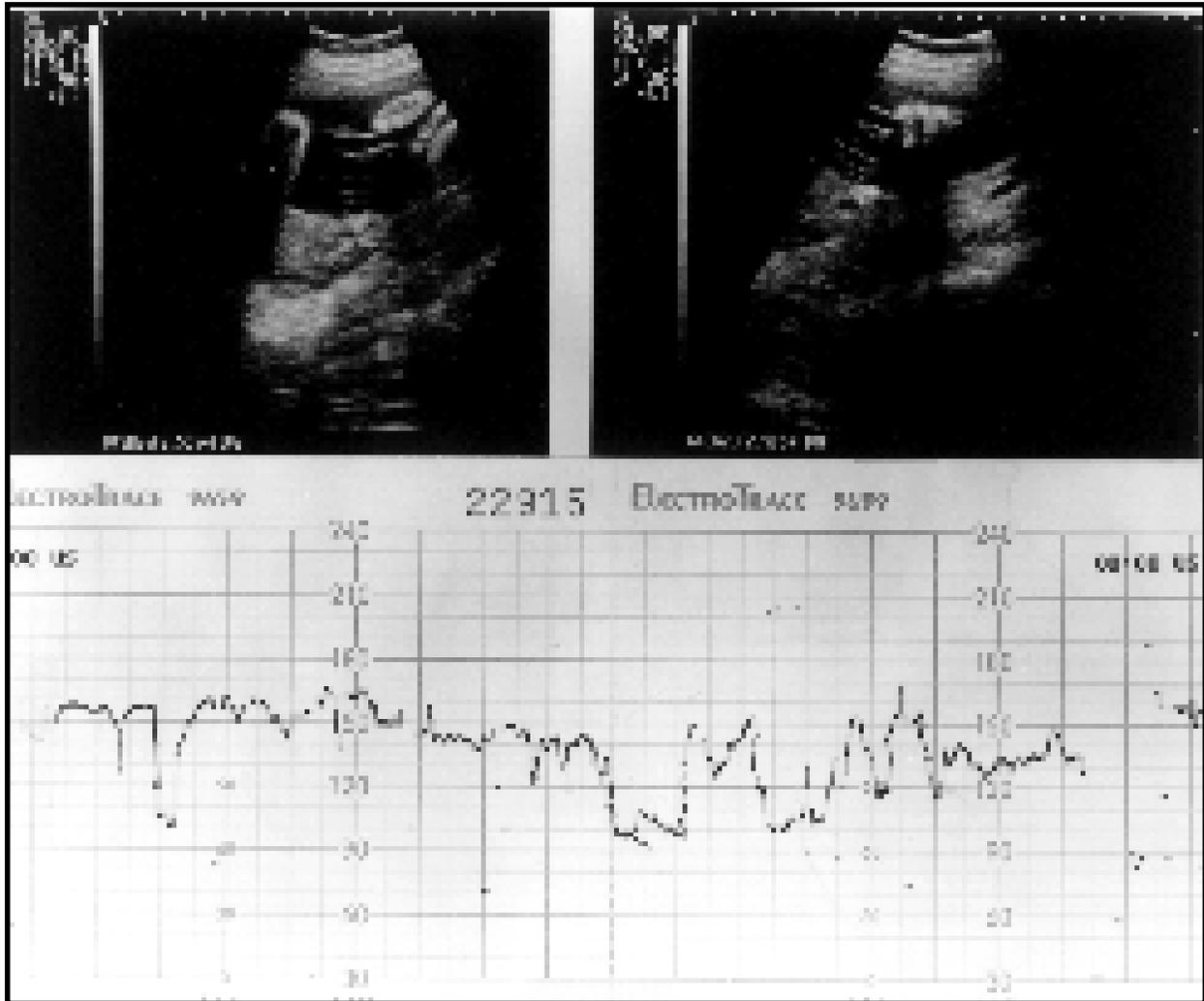
Long Umbilical Cords: As previously described, a long cord would be considered greater than 70 cm, although 90 cm has been used as the measurement. Perhaps a long cord in an abnormal sense should be defined as the length of cord which can cause fetal harm by entanglement. If so, such a cord would be 4/5 as long as the fetus. Long cords become a problem when circumstances predispose the cord to prolapse (slip out of the womb) and are more likely to prolapse simply because there is more of it.

In addition, the umbilical cord is heavier than amniotic fluid and therefore sinks. When visualizing the cord with ultrasound, a cord may seem “suspended” in the amniotic fluid because of hydrostatic pressure and position. It may appear to float at the top of the uterus, depending on the mother’s position. Some papers even comment that the cord “floats.” Yet the cord does not float; it sinks. The cord can find its way toward the cervix. It always falls away from the fetus even if the fetus chooses to handle it. This sinking property of the cord may be important in preventing cord entanglement. It may be nature’s way of reducing the risk of the fetus harming itself.

The cord usually bunches up in front of the fetus, commonly near its legs and feet. Long cords may make themselves more available to the fetus and enable the fetus to “mouth” the cord like a pacifier, or handle the cord like a toy. Whether or not this is preferable depends on whether cord blockage is present during the sucking and handling. These simple fetal behaviors may play an important role in fetal well-being and development. Reflexes may exist which serve the purpose of short duration cord play.

How hard a fetus can squeeze its fist at a given gestational age is unknown. Knowing whether the fetus can grab the umbilical cord and obstruct its own blood flow would be helpful. The grasp would have to exceed its own blood pressure and squeeze with a pressure greater than the fetal systolic pressure (60 mmHg). Could the strength of some fetuses be a disadvantage? Could the thickness or thinness of cords predispose the cord blood flow collapse if squeezed? Does the fetus faint and let go? Some anecdotal reports suggest that all these scenarios are possible. (Figure 16)

A long cord in and of itself is not dangerous. What becomes dangerous is the way in which the fetus interacts with its supply line. “The Collaborative Project,” a national study of over 55,000 pregnancies, concluded that long cords are associated with fetal entanglement. The key here is to understand that entanglement depends on fetal repositioning in the uterus.



(Figure 16) Fetal cord grasping as seen on ultrasound, with fetal heart rate pattern

Does the umbilical cord itself cause the problem, or does the fetus have to cause the problem? What does the fetus do to become entangled? Are there identifiable factors contributing to an active fetus (one which frequently repositions itself in the uterus and increases its chance of cord entanglement)? Lastly, what role does the mother play when she is active, and does diet play a role? Dietary factors and fetal behavior are currently being investigated. Maternal exercise is also being studied relative to its effect on the fetus. Fetal behavior over time is an active area of research. Scientists are looking for patterns which could mean the difference between well-being and illness.

“A better understanding of the physiology of pregnancy and labor has done away with the theories that the activity of the mother causes coiling of the cord.”

John Paterson Gardiner, M.D.
Toledo, Ohio, 1922

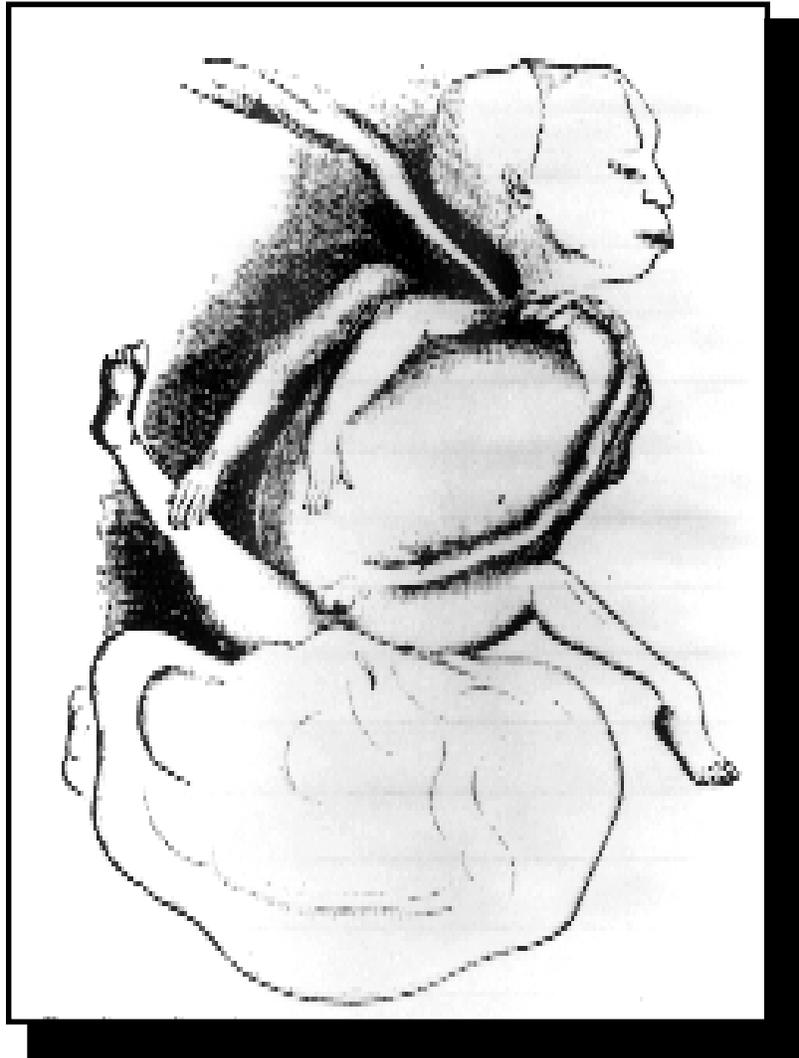
Nuchal Cords: When a fetus' umbilical cord crosses itself 360 degrees around the neck, it has a nuchal cord. Dr. J. Selwyn Crawford of the British Medical Research Council first defined this problem in 1962 as “the condition in which the umbilical cord is wound at least once around the neck of the fetus.” This initial description was applied to a study which made the following conclusion: “[the] nuchal cord is well recognized as being commonly associated with fetal distress and neonatal depression. It is all the more remarkable, therefore, that little work has been published to demonstrate the incidence of the condition, and to analyze its effects during labour and delivery.”

J. Selwyn Crawford, M.D.
British Medical Research Council
1962

This finding was observed in 15% to 30% of nuchal cord deliveries. (Figure 17)

Great variances of nuchal cord observations exist because prenatal diagnosis with ultrasound has only recently been made. The accuracy of previous studies is in questions because close adherence to specific definition may not have been followed. Nuchal cords are at the center of the supply line controversy because so much differing information exists. To study nuchal cords, the fetus involved must be prenatally identified. Cords can wrap around the fetal neck as soon as the cord reaches 4/5 of the fetal length. This occurs as early as 10 to 12 weeks gestational age.

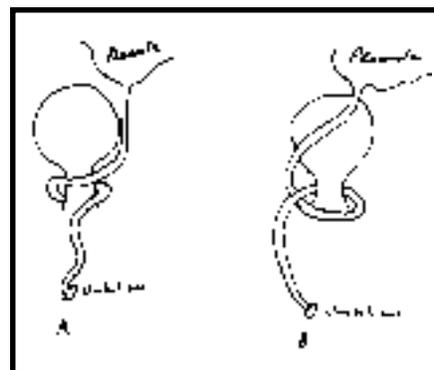
Fetuses can entangle themselves multiple times; deliveries of fetuses with 6 to 8 complete



(Figure 17) Fetus with tight cord around the neck - French drawing

loops around the neck have been described. What distinguishes a surviving fetus from a stillborn fetus with a nuchal cord is unclear; however, some insights exist. For example, fetuses born dead with cords around the neck have been described from abortuses (8-12 weeks) to postterm (42-44 weeks). Yet no one clearly understands when these fetuses die, and no one has clearly observed those cord patterns.

Actually, not all nuchal cords are the same. One researcher noticed a predisposition to nuchal cord related deaths around 38 weeks. This is difficult to verify because ultrasound was not used to accurately date the fetuses. Another researcher noticed that two patterns exist: the cord crosses over itself, or the cord crosses under itself. (Figure 18) This observation may be important when considering knot formations in the cord. A cross-under pattern is needed for knot formation and can cinch a nuchal cord if it rolls back on itself.



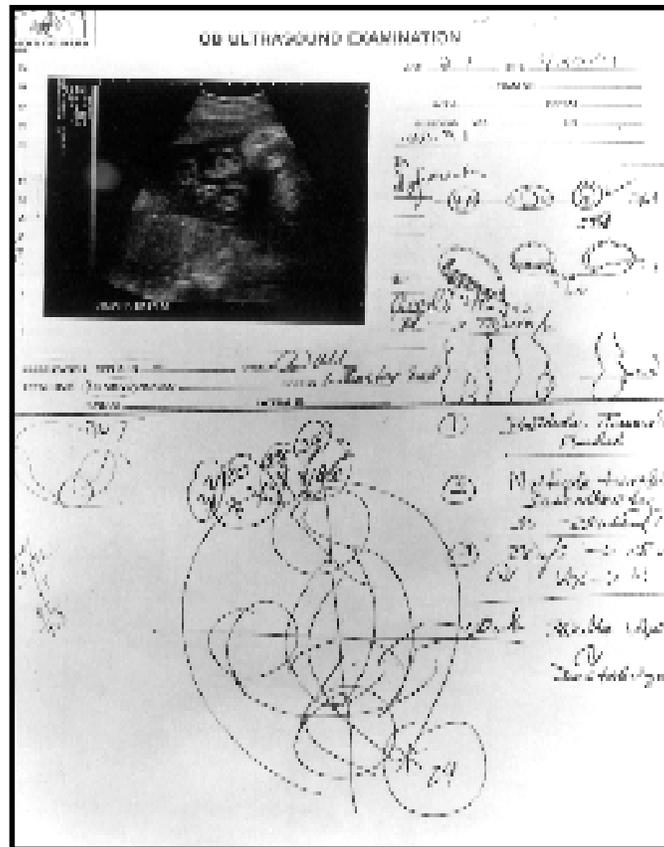
(Figure 18) Dr. Giacomelli described two types of nuchal cords, A & B

Are multiple nuchal loops more deadly than single nuchal loops? Does the chance of fetal death increase with the amount of loops in the cord? Again, a definite answer is unknown, but many factors have been noted. Regardless of the number of loops, the problem lies in supply line disruption. For example, fetuses who are breech are more likely to have more loops than fetuses who are vertex (head down). The shoulders of breech fetuses are freer to rotate than those who are vertex. Placental position is another factor. Activity, again, may influence the formation of nuchal loops. In addition, male infants seem more predisposed than female infants. There are also combinations of these factors.

Nuchal cord formation is a function of fetal life. Once a cord is “long,” it is more likely to affect an active fetus. A very active fetus, especially one with a high level of amniotic fluid volume and a high level of energy derived from placental design, may be predisposed to nuchal cord formation. (Figures 19 and 20)

The number of infants killed by their umbilical cords is unknown. The National Center for Health Statistics does not have enough data to determine this. Possibly as few as 4,000 deaths per year or as many as 8,000 deaths per year involve umbilical cord complications. The important point here is these infants are normal; they are normal, but they are dead.

Some researchers believe that cord accidents do not cause death. However, Dr. Arnold Lillien explains this may not be correct.



(Figure 19) Research notes diagraming fetal repositioning



(Figure 20) An 18 week fetus with a double nuchal cord - as seen on ultrasound

“In general, however, the consensus of opinion has been that a nuchal cord is unlikely to compromise a fetus and is rarely, if ever, a cause of fetal death. We found a significant incidence of tight nuchal cords among term intrapartum fetal deaths without explanation.”

**Arnold A. Lillien, M.D.
Perinatal Research Branch
National Institute of Health
1970**

A similar opinion is shared by Torgrim Sørnes, M.D., who has studied this issue for two decades.

“The pathology of umbilical cord encirclements around the fetal body, neck, or extremities has not been subjected to thorough study for years, and little is known of their etiology, pathogenesis or effects on the fetus.”

**Torgrim Sørnes, M.D.
Department of Gynecology and Obstetrics
Akershus Central Hospital
Nordbyhagen, Norway, 1995**

Cord Entanglement: The umbilical cord can surround an extremity, the body, or the neck. Body loops can be single or multiple and can exist with nuchal cords. The number of body loop incidences is unknown. Most of these entanglements are undone at delivery as the infant is being born; therefore, they are never witnessed and never recorded.

The effect of a body loop is cord compression. Tight loops have made impressions on the skin of the fetus and can restrict fetal movement in the uterus. Loops around the extremity can affect circulation of the extremity and cause damage to a foot or a hand. Circulation disturbances can sometimes form blood clots in the arteries, vein, or placenta. These events can change the oxygen supply to the fetus and cause growth disturbances or death.

Yet the variety and magnitude of cord entanglement becomes evident when one considers factors such as cord design, Wharton’s jelly thickness, placental types, cord insertion sites, and fetal position. The explanation is not impossible and may be finite. There are no prenatally derived

statistics which can provide the scope and incidence of cord entanglement. To arrive at this explanation, an understanding is needed of the number of prenatal incidences, the length of time that non-nuchal cord entanglement lasts, and the effects on the fetus.

Combinations

“These experiments show that even a slack knot may be sufficient to interfere with, if not completely obstruct, the cord circulation, but that any pull upon the knot such as might be exerted if the cord were wound around the child’s neck or body as to cause a relative shortening of it, would easily cause sufficient tightening to impede the circulation completely.”

Francis J. Browne, M.D.
Edinburgh Royal Maternity Hospital
Great Britain, 1923

In addition to individual cord conditions creating fetal disturbances, potentially dangerous combinations are often observed. Yet the risk of harm is unknown. Whether a combined cord complication increases the chance of stillbirth compared to a single cord complication is unknown. (Figures 21/22). Another unknown is the effect the mother’s toxemia or a disorder such as anemia may have on the fetus.

Combinations involving fetal umbilical cord insertion site hematomas (vessel rupture) have been observed. These hematomas were associated with nuchal loops, true knots, and mid-cord hematomas. These particular combinations suggest an umbilical cord subject to stretch mechanisms, possibly tearing the umbilical vein near its insertion.

Reports of infants born with more than one knot and as many as three imply that fetal activity and repositioning are key factors. Were all three knots formed at once or one at a time? Is there a time during fetal development which is at highest risk for multiple knot formation? The gestational age at which knots form would be valuable information so that increased screening could be initiated at that gestational age.

Infants delivered with nuchal cords and a true knot are not uncommon. Upon closer inspection, the cord is usually long. The excessive length increases the probability of knot formation, but it is not necessarily deadly. Increased slack decreases the chance of tension. Cord architecture is also a factor. However, in studies that recorded length, cord type was not included.

(Figure 21) Ante partum fetal death due to vascular strangulation. The two funicular coils around the neck and the true knot in the lower coil are well visible.



(Figure 22) Effect of pressure of umbilical cord round the leg and neck

Torsion: Torsion is the condition of the umbilical cord where twists are superimposed on the cord itself. Knots and nuchal cords are not always seen with torsion, but they can be observed with torsion. This combination, though, can be dangerous because cord positions that allow tension will cause torsioned cords to kink. Torsion is tolerable as long as the torque (imposed twist force) is released as a deformation of the cord (a snarl). (Figures 23,24,25) If the energy of the snarl is re-imposed, the cord blocks just like a garden hose. A deadly combination is a cord under torsion with a shoulder loop, nuchal cord, body loop, or extremity loop.

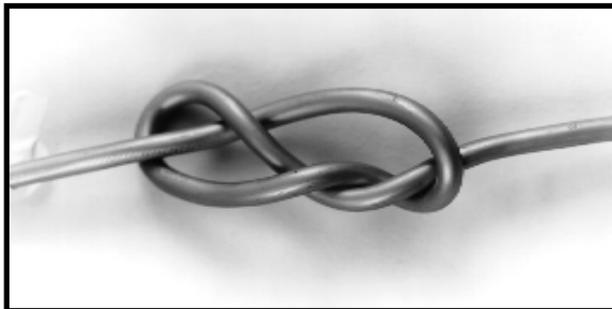
Finding vessel thrombus combined with torsion is not unusual. These thrombi can also be



(Figure 23) A ten week fetus with natural helices counted on ultrasound



(Figure 24) Umbilical cord torsion seen with ultrasound - no helices



(Figure 25) A model of a snarl becoming a knot

seen in the surface vessels of the placenta.

Many of these combinations go unnoticed simply because no one is looking for them. When blockage occurs with cord compression mechanisms, one of the clues left behind is edema (a build up of fluid) of the cord. It is not unusual, for example, to have a nuchal cord with proximal cord edema suggesting mild episodes of blood flow disturbance but not enough to cause death. In

addition, it is not unusual for these combinations to cause fetal heart rate changes severe enough during labor to cause an emergency C-section.

Short cords may be more dangerous because they are shorter, may marginally attach and may be a velamentous insertion. This increases the chances of umbilical cord rupture compared to a normal insertion. Short cords may also predispose the cord vessels to thrombosis, hemorrhage, or hematoma formation. Individual reports of such occurrences are published in the medical literature. Although not common individually, all combinations as a whole present a significant occurrence when viewed on a larger scale.

When combined, these events represent a more prevalent failure of reproductive mechanisms when compared to the most common genetic malformations. Altogether, in any community hospital practicing obstetrics, cord complications will be observed more often than congenital defects. For instance, one medical review of just “hematoma” formation of the umbilical cord at Johns Hopkins Medical Center was observed in one out of 5,505 cases. Mongolism, a common genetic alteration known as Trisomy 21 Syndrome, occurs overall 1 in every 660 deliveries. Trisomy 18, another readily acquired genetic defect, occurs 1 in every 3,000 deliveries. Trisomy 13 occurs 1 in every 5,000 deliveries.

Hematoma of the Umbilical Cord

“The causes of rupture of an umbilical vessel are obscure and probably several factors enter into the development of a hematoma in each case.”

A. Louis Dippel, M.D.

Johns Hopkins University and Hospital

Baltimore, Maryland, 1940

A hematoma of the umbilical cord is due to bleeding into the substance of the umbilical cord. They can be spontaneous, iatrogenically induced, traumatic self-induced, or secondary to an umbilical cord defect. The usual risk of cord hematomas that is often quoted is 1/1,000 to 1/5,500 rps deliveries. Hematomas can be due to the umbilical artery or umbilical vein. This sausage-shaped injury seems to be more frequently noticed on the fetal end than on the placental end. The chance of death to the fetus is as high as 50% and as low as 14%.

The cause of hematoma development is unknown. Some scientific insights suggest “wearing” of the vessel walls, thinning of the walls, tearing, and then bleeding into the substance of the cord. Compression of the cord vessels leads to clogging and then death of the fetus. The sight of

hematoma formation can vary however.

Again, unknowns exist in the area of umbilical cord hematomas. For example, it is unclear whether the cause of the fetal end hematoma is the same as the placental end hematoma. The most common time of hematoma formation is also unknown. Most discussions of formation have centered around labor and delivery. This would suggest that cord tension may play a role, and this tension can possibly be attributed to a condition where the cord is stretched. (Figure 26)

Obviously, observation of this condition was recognized long before recorded history and must have motivated many speculations. Since most modern reports (1871 to present) have described mostly stillborns, cord compression is an end result.

Numerous descriptions focus on hematomas which originate from the umbilical skin. Vessels penetrating the Wharton's jelly from the umbilicus may rupture from fetal manipulation which includes pulling on the umbilical insertion. Infants can grasp before birth and have been observed sucking and pulling on everything in the uterine amniotic cavity.

The most difficult feature of umbilical cord hematomas is that they are spontaneous and evolve quickly. Many case descriptions suggest that the fetuses were not in labor. More information is needed about the timing of these events and particular fetal behavior that may predispose the cord to hematomas if the fetus is vulnerable.

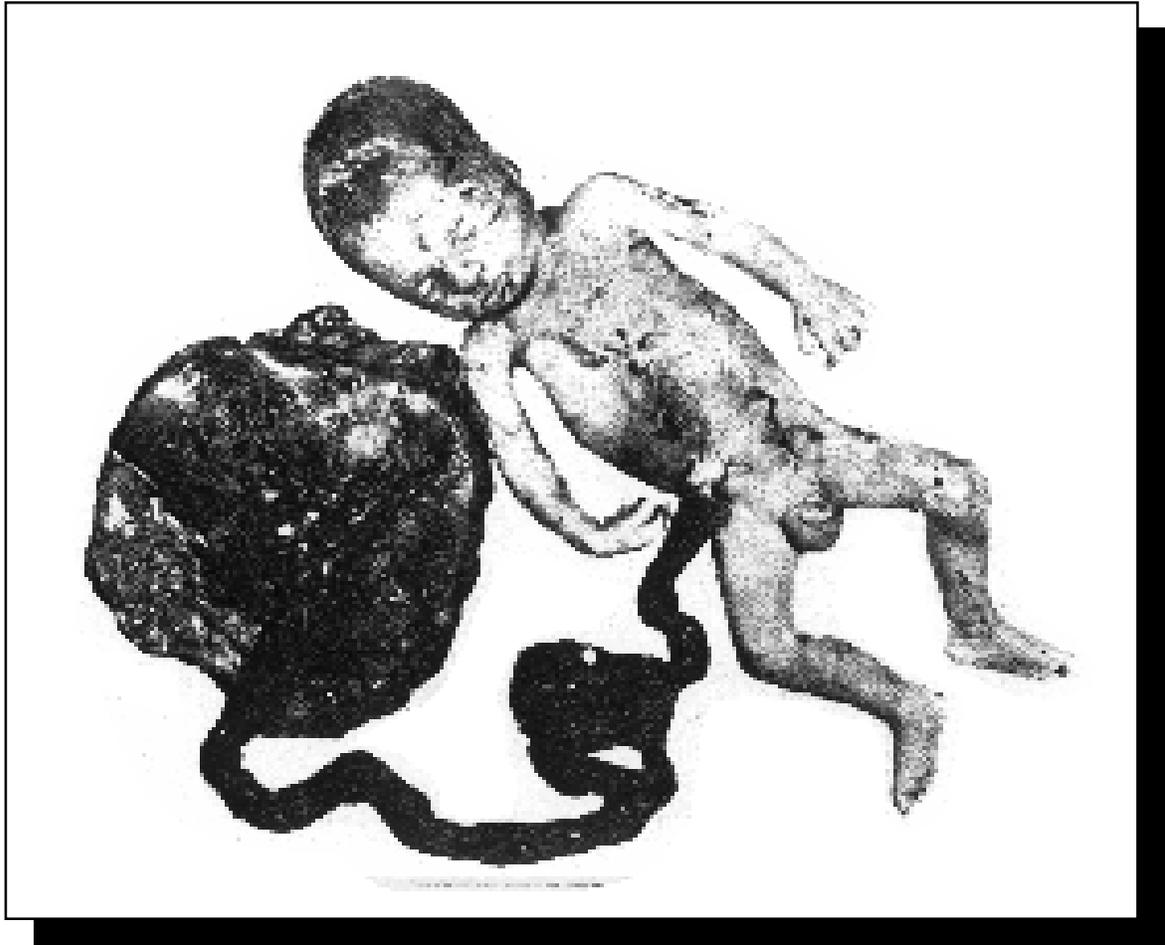
Torsion: As previously described, torsion is the condition of the umbilical cord where twists are superimposed on the cord itself, similar to an overly twisted telephone cord. Umbilical cord torsion may not be an unusual finding.

“In human beings, the umbilical cord is subject to many kinds of torsion, coiling, looping and knotting during pregnancy or during parturition. Torsion of the cord is a common occurrence.”

W. G. Atwood, M.D.
Fall River, Massachusetts
1932

No formal definition of torsion currently exists. Veterinary scientific literature is quite familiar with the problem of torsion, especially in thoroughbreds. In fact, torsion is often a cause of fetal loss in horses. Veterinarians readily observe this finding and look for confirmation by studying other fetal structures confirming its presence as the cause of death. In one study of fetal losses in horses, 200/2,000 were due to this umbilical cord accident.

Another topic more easily discussed in animal research is the association of torsion with



(Figure 26) Umbilical cord hematoma with rupture of a vein varix.

heart failure and the finding of thromboses in the placenta. Clearly torsion is readily accepted as a cause of fetal death in animals. For humans, this is not the perception. Some think that torsion occurs after death as a result of random movements of the dead fetus.

Prior to the Pregnancy Institute's study of torsion of the umbilical cord, no case report of a liveborn with torsion existed in the medical literature. All descriptions of human umbilical cord related torsion have been in stillborns. Therefore, the Pregnancy Institute's information did not come about easily. Dr. Kurt Benirschke, a Harvard-trained pathologist and Professor Emeritus of Pathology at the University of California, San Diego, has provided us with invaluable insights into the mystery of this fetal/placental abnormality, especially torsion of the umbilical cord. He determined that the cord becomes intensely twisted, placental damage occurs secondary to thromboses, a common time of death is between 28 and 30 weeks gestations age, and torsion of the umbilical cord is seen in cerebral palsy cases.

With this information, our observations of umbilical cord torsion began with normal live deliveries. Dr. Benirschke's suggestion of an intensely twisted umbilical cord prompted us to untwist the cord in over 400 deliveries. We determined that torsion is actually very common, but not always pathologic. Next, we determined the time in which pathology occurs and the spectrum of effects. The effect of torsion can be fetal harm ranging from heart failure to stillbirth.

Torsion is not a natural state of the umbilical cord. Torsioned cords must not be confused with naturally "helical, coiled, or spiralled" cords. The appropriate terminology should be the word "helical," which implies a constant, if not same diameter. Torsion and natural helixes are different from each other. According to European literature, the curly course of the cord was considered in 1521, and left versus right (clockwise/counterclockwise) patterns were recognized in the 1600s. Left curls (80%) predominate over right curls (20%). Early estimates of total curls averaged 11 turns per cord (Hyrtl 1890). These estimates may be different from each other, however, because twists were not removed by unwinding. Most umbilical cords on the average (55 cm) have 3 to 5 helixes, the upper limit having 8 helixes. The average number of twists is 3. Together, the average curls could be Hyrtl's 11. These same quantities are also found in equine cords. Cords can become twisted when the fetus repositions itself. Therefore, torsion is not an umbilical cord anomaly or abnormality. It is a mechanical deformation/alteration due to the behavior of the fetus.

The importance of the formation of torsion implies much about intrauterine life and the dangers which face the fetus. These dangers are as significant as those which a newborn gazelle faces on the African savannah. The fetus begins to move around 10 weeks gestational age. If fetal activity is excessive, trouble begins.

Torsion, nuchal cord formation, body loop formation, and true knots are related. The relationship depends on the type of repositioning which takes place. Fetuses can tumble, roll, and somersault like a gymnast in the three dimensional environment. Torsion occurs when the fetus imparts these motions to the cord. This mechanical energy is called torque and is subject to the same laws of physics as any structure that is deformed by the same force. Twists occur as a result of this torque, and torsion occurs when twists involve every ninth centimeter or less of cord length. Torsion becomes deadly when it reaches a ratio of one twist per five centimeters or less of cord length.

Torsion of the cord is a remarkable example of nature on the edge. For example, if a 50 cm cord has 10 twists imparted to it by 30 weeks, it can relieve the torque by growth of the cord. If the fetus remains in one position after 30 weeks, the cord could grow another 10 cm and change from a ratio of one twist to 5 cm (1/5) to one twist to 6 cm (1/6). The danger is therefore averted.

Collapse and kinking of the cord blood vessels seem to occur at a ratio below 1/5. The cord must then stretch so it can redistribute the stress/strain of the torque, which usually ends up being at a point near the placental end of the cord. A simple example is a telephone cord. Once heavily twisted, it narrows and snarls (loops over itself). Over time it becomes a complete mess. (Table 3 and Figure 27)

Torsion is an umbilical cord complication which is dependent on a number of factors. For example, fetal behavior is determined by intrauterine needs. This intrauterine fetal ballet has a purpose, function, and plan: get the fetus into a comfortable head down, pre-labor position. The fetus may attempt this several times before reaching the “milestone.” Its success within a few attempts depends on placental position, amniotic fluid volume, inner ear (vestibular) maturation, and maternal diet and activity. If one of these factors is exaggerated, the fetus may begin the process of excessive activity, continued repositioning and torque of the umbilical cord.

Because torsion and its effects are also dependent on umbilical cord length, short cords may succumb faster than longer cords. Again, the type of cord may also play a role where Type IV (vein around arteries) may be more prone to collapse under equal stress than other types (arteries around vein).

Cord diameter/circumference also plays a role. A thicker cord may offer more resistance to torque than a thin cord. This is in need of study and will require a specialized vascular laboratory.

Fetuses may also possess natural reflexes which allow them to untwist themselves. Cord helixes can be left and right in nature, mostly left (80%). If the fetus counterpositions itself, under favorable conditions of intrauterine life it can get itself out of a perilous torsion.

How often and in what way the fetus repositions itself during intrauterine life are unknown. One clue to this question is the twists seen at delivery. We have found that the average number of

Table 3
UMBILICAL CORD CHARACTERISTICS ASSOCIATED
WITH TORSIONED AND NORMAL CORDS

Patient	Weeks Gestation	Total Curls	Cord Length	Twists	Twist Ratio	Non-Stress Test Fetal Heart Rate	Placental Location
A	28	26	65	15	4.3	Fetal Death	Posterior
B	30	12	30	7	4.3	Fetal Death	-
C	30	15	42	10	4.2	Premature Labor	Posterior
D	37	21	60	12	5.0	Decelerations	Posterior
E	39	24	55	11	5.0	Decelerations	Posterior
F	37	28	90	16	5.6	Decelerations	Anterior
G	38	22	70	12	5.8	Variables	Posterior
H	30	14	38	6	6.3	Premature labor	Posterior
I	40	20	65	10	6.5	Variables	Posterior
J	40	32	95	14	6.8	Not Tested	Posterior
K	39	24	70	10	7.0	Variables	Posterior
L	36	12	55	7	7.9	Decelerations	Posterior
M	39	21	95	12	7.9	Variables	Anterior
N	40	16	75	6	12.5	Normal Pregnancy	-
O	40	17	80	5	16.0	Normal Pregnancy	-
P	41	12	65	4	16.3	Normal Pregnancy	-



(Figure 27) Umbilical cord torsion with mid cord narrowing.

twists is 3 1/2. This was determined by untwisting over 300 umbilical cords at birth. It suggests that the fetus repositions itself in one direction 360 degrees (3 1/2 revolutions). Unfortunately, the fetus may move in clockwise and counterclockwise directions. This would suggest that six moves left-to right could be negated by three moves right-to-left, leaving the three twists as an inaccurate record. The fetus probably moves more often in the same direction and usually does not reposition itself often. Ideally, a constant series of ultrasound exams or a device which detects fetal position (like global positioning systems) might give us the final answer. Knowledge of the usual intrauterine activity over time would help solve the problem of torsion. [Insight - skin reference which would be trackable.]

Umbilical Cord Knots:

“The number of ‘knots’ on the umbilical cord of a first born child is held to foretell how many of a family the mother is to have.”

“Umbilical Cord Folklore”

The British Medical Journal

1912

On first inspection, it is remarkable that a fetus can tie a knot in its umbilical cord. How is that possible? Monoamniotic twins (twins in the same sac) can create multiple knots together. No reports, however, exist that siamese twins had knots in their common cord. This twin anomaly of reproduction suggests that the fetus must move to form a knot. Two separate fetuses moving independently create cord chaos which can lead to multiple sites of constriction and fetal death. So how does a knot form, and what happens to the knot in utero?

Most true knots are probably not formed before birth but during birth. It is highly likely that the fetus is entangled with the cord and, when delivered, is pulled through a loop of cord, forming a knot. To form a knot, the loop of cord must be special. It must be long enough for the fetus to form a nuchal cord. The nuchal cord must cross under itself, not over itself. This cross-under pattern allows the formation of a single hitch knot. The loop of crossed-under nuchal cord must pass over the fetal body before a knot can form. This specialized loop sometimes forms around an ankle and entraps it. The fetus is born with a snared ankle instead of a true knot. Sometimes both ankles can be involved. If the knot completes itself, the risk of blockage is great.

We speculate that prenatal knots may form prior to 32 weeks, a common time of fetal repositioning. Yet the time at which most knots form prenatally is unknown. Knots may form

earlier and have been noted prior to 20 weeks in miscarriage specimens. The earliest knot seen prenatally with ultrasound is in monoamniotic twins at 19 weeks.

To envision knot formation, it is important to remember torsion. As the fetus repositions in the uterus, it applies torque to the umbilical cord; this energy causes the cord to loop counter to the direction of the torque, thereby crossing counter to the fetal movements. A cross-under loop has now formed which, if lassoed around the fetal neck, can work its way along the fetal body to form a knot. The fetus may not even have to change position because its movements may work the loop to its feet and then off. (Figure 28)

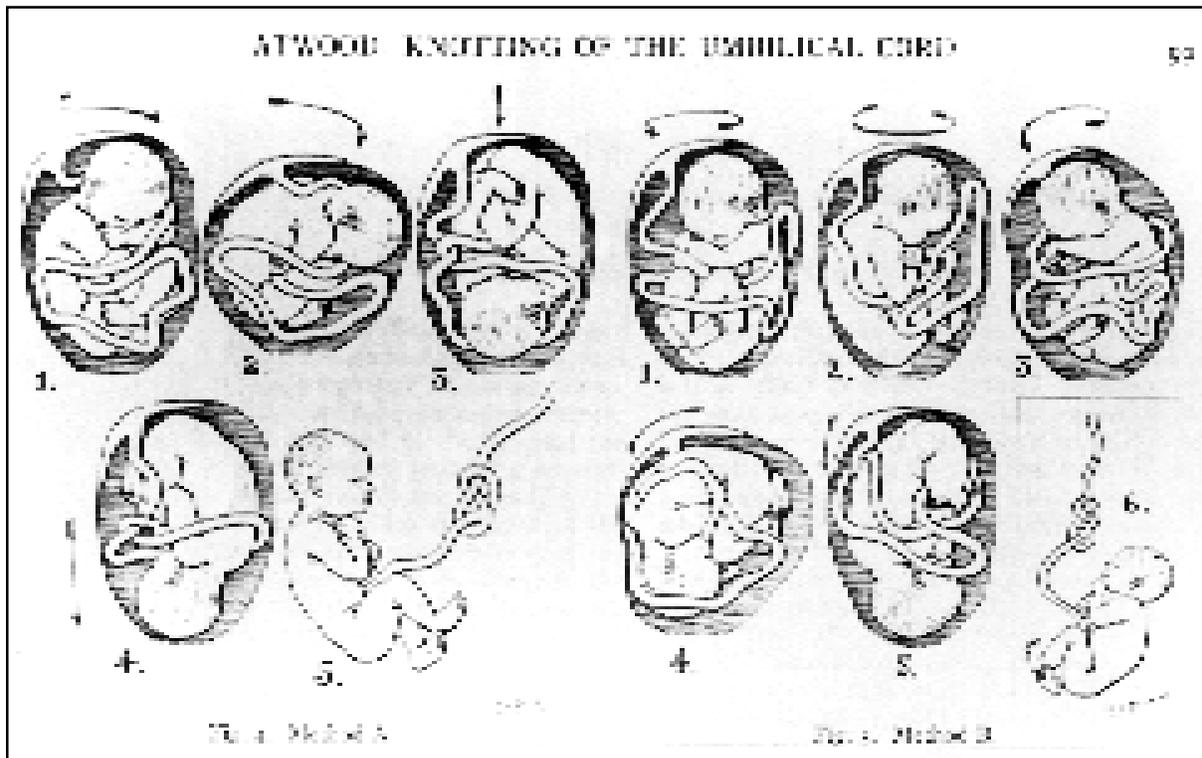
Knot complexity depends on the amount of snarls created at the base of the loop. One snarl creates a single knot, two snarls a double knot, and three or more snarls a complex knot. Double snarls with double loops have also been described. The whole process is actually simple. A complex knot does not imply multiple fetal tumbles and twists. It only means the fetus passed through a loop or loops simultaneously.

How the fetus can cinch a knot tight and block blood supply depends on other factors. As with a prolapsed cord, complete compression of blood flow will cause fetal harm. A knot can completely block the cord, and a knot can be tightened by the fetus during prenatal life. The chance of having a fetus deliver with a true knot of the umbilical cord is on the average 1% to 2%. The chance of fetal demise secondary to a knot blockage is 5% to 10%. It is not known whether the most common knot is a single, double, or triple knot. It is also unknown whether more complex knots are more deadly. It is known, however, that the chance of fetal death is increased with a knotted cord.

Body/Extremity Loops: Body and extremity loops, one of the more difficult mechanisms to observe, may be a significant cause of fetal decompensation. Anecdotal reports have described extremities damaged by tight loops. The ability of these loops to injure an arm or leg but not cause fetal death is difficult to determine. Stillbirths are observed with multiple loops around ankles, necks, and bodies; yet it is difficult to determine which compressed segment caused death.

Cord loops around the body are usually not observed because delivery unwraps them. The "loop puzzle" is pulled apart at the moment of delivery, so it goes unseen. The incidence seen at delivery is probably less than what actually takes place prenatally over time; probably 0.5% to 2% is reported. The increased chance of stillbirth is still debated.

Chinese and Russian medical literature tend to suggest that loops are not benign. If a fetus persists with a body loop, the chance of cord compression appears greater than if no loop exists. Body loops also can act like wenches, taking up slack and causing relative shortness of the cord. This shortness may lead to placental separation and, in one report, maternal amniotic fluid embolism.



***(Figure 28) Dr. Atwood's Animation of knot formation theory: (Double knot) 1930.
(see also Figure 21)***

Does a fetus recognize that its supply line is being compressed? Is there a fetal instinct which reacts to limitation of movement? Currently, some basic science experiments in rats suggest that this exists. Observations of entangled fetuses suggest the fetus is moving in one direction and not back and forth. Unwrapping a triple nuchal cord is usually in the same clockwise or counterclockwise direction in which it formed. Most loops originate over the fetal right shoulder and unwind (counterclockwise). Similarly, torsion untwists clockwise. Fetuses may instinctively be pre-programmed to register favorable right to left “rolls”.

Whether this is totally reflex, chance, or instinctive evasive movements is unknown. The role of the inner ear (balance, orientation) vestibular system is also unknown. Could the fetus become “faint” or “dizzy” in utero? If so, does this cause unusual fetal movements which lead to tumbles/somersaults and rolls? Can diet play a role? It is suggested that caffeine and xanthines (tobacco, chocolate, tea, coffee, soft drinks) may keep the fetus awake. Does this predispose the fetus to more activity and possibly more risk?

Does the fetus develop a sense of position as well? After all, fetal hearing matures around 24 weeks. The fetus hears low frequency sounds. The vestibular system is an ancient, evolutionary sense. Fish have had it for a long time. Is the fetus using its well-developed sense of position, since its taste, smell and sight senses cannot determine its orientation? If so, can its sense of position be disrupted by other stimuli and send it into a search pattern of disoriented movement resulting in body loops? How cord compression affects the fetus is discussed in the next chapter. How the fetus behaves and why it may be designed not to entangle itself will also be addressed next.

“It is suggested that the reduced fetal movements and the changes in fetal heart rate were due to a diminished blood flow in the cord vessels as a result of gradual cord compression.”

E. Sadovski, M.D.
Jerusalem, Israel
1976

“Three umbilical cord complications occur at least once in every four deliveries, and their problem cannot be avoided.”

W. N. Spellacy, M.D.
Minneapolis, Minnesota
1966

Chapter 3: Fetal Behavior and Physiology

“Whatever may be the final determination concerning the etiology of coiling of the cord, the movements of the foetus will undoubtedly play a prominent part in it.”

John Paterson Gardiner, M.D.

Toledo, Ohio

1922

“Any fetus that frequently changes position as found by routine examination is a candidate for torsion, looping, or knotting of the cord.”

James P. Hennessy, M.D.

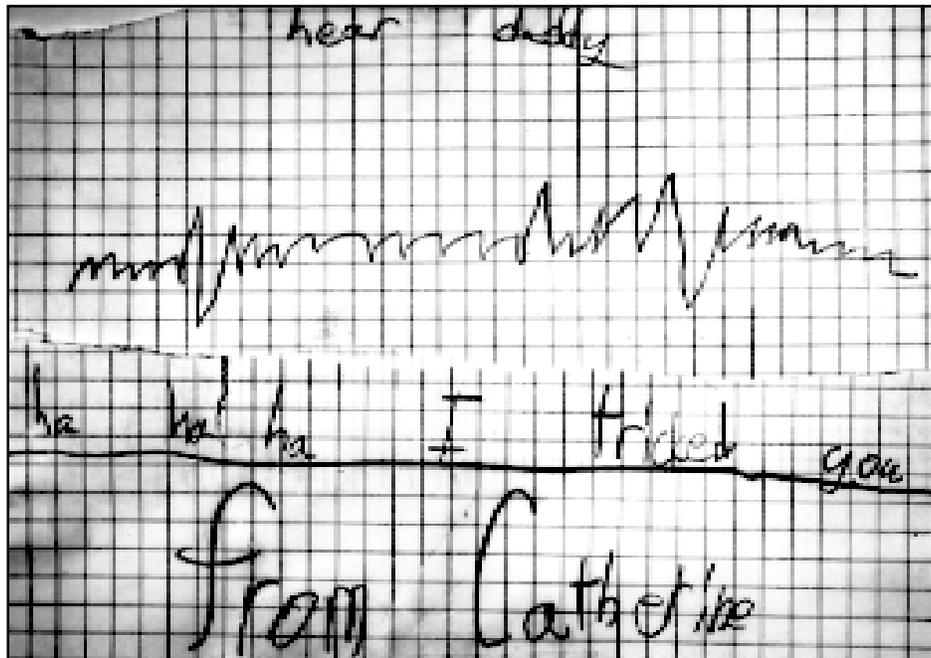
New York, New York

1964

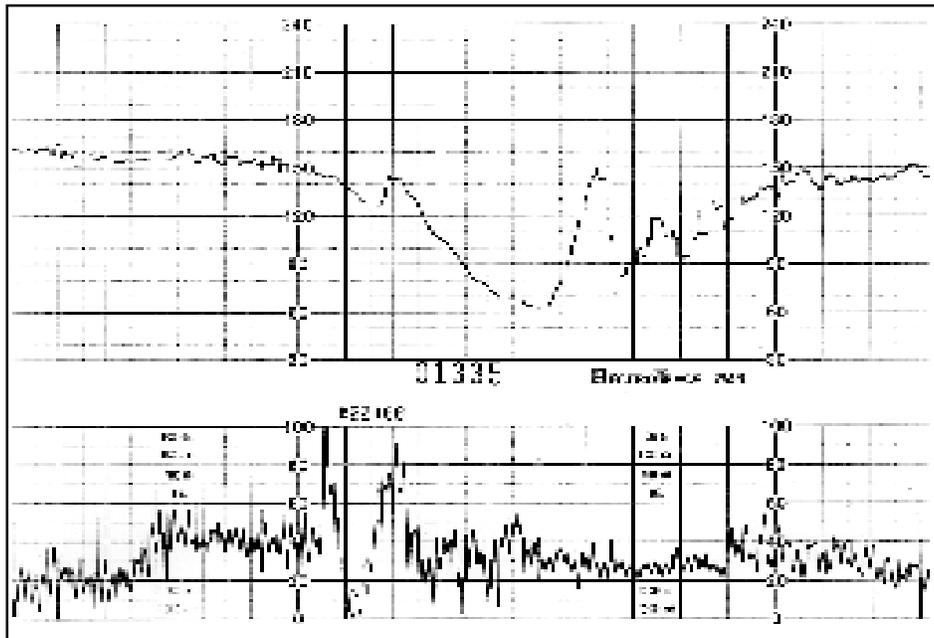
Before the invention of ultrasound technology, much uncertainty surrounded the unseen developing fetus. How the fetus grows, plays, sleeps, and acts in the uterus was not certain. Ultrasonography allowed visualization of the fetus inside the mother as it transforms itself up to the moment of labor and delivery. Visually studying these behaviors has allowed for new insights.

Fetal heart rate monitoring is the detection and recording of the fetus’ beat-to-beat heart rate. This non-invasive electronic tool allows study of fetal reaction to its intrauterine environment under stressful conditions. Changes in the quality and character of the beat-to-beat pattern of the fetal heart allow graphic patterns to be recorded which translate into some basic fetal physiologic conditions. Fetal heart rate monitoring and ultrasound imaging together have contributed to the overall impression obstetricians have today of how the fetus works and interacts with the placenta, umbilical cord, uterus, and mother. (Figures 29 and 30)

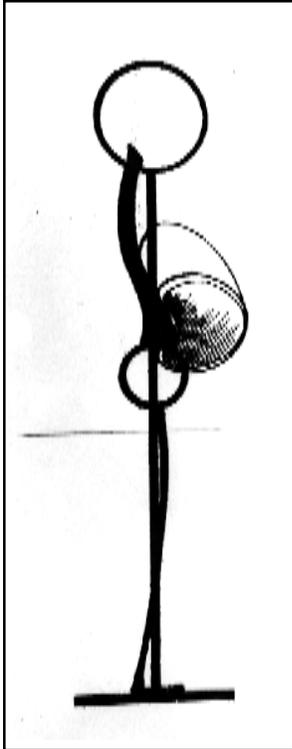
Dr. Peter Nathanielsz, M.D., Ph.D., has studied fetal development for the National Institute of Health. He states, “Ultrasound shows us that each fetus has his or her own personality. — We can use ultrasound as a video camera on the developing fetus. We can track the fetus’ heart beat and can follow his responses to sound and uterine contractures (uterine cramps which last 5 minutes).” Ultrasound reveals the intricate systems which have evolved to allow human survival and continu-



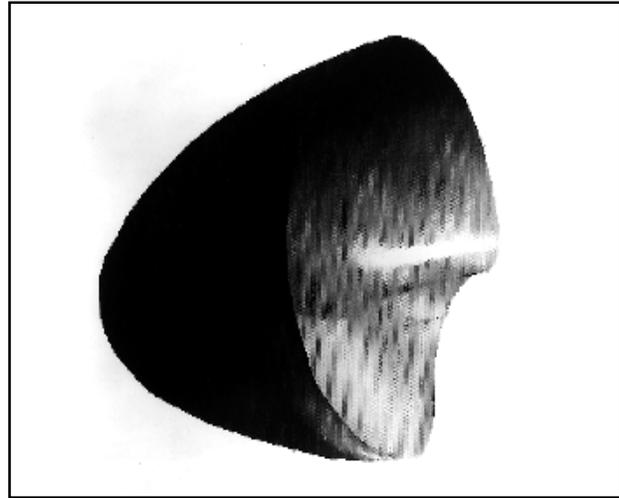
(Figure 29) A child's view of a fetal heart rate with variables.



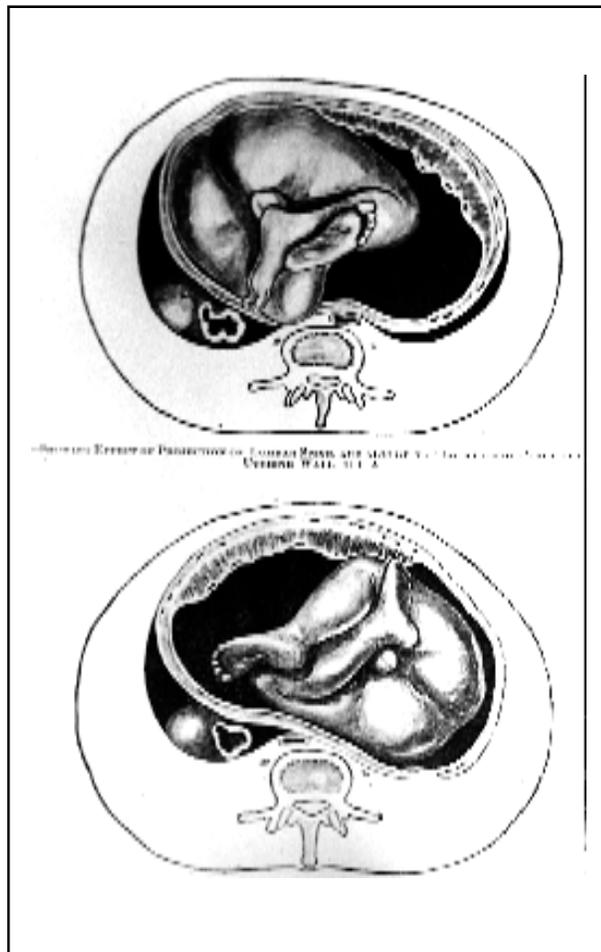
(Figure 30) A fetal heart rate pattern showing deceleration to 60 BPM with recovery to 150 BPM.



(Figure 31)
Cordosis and the
uterus.



(Figure 32) **Three dimensional view of the uterine**
cavity.



(Figure 33) **Two dimensional uterine view with fetus**
from Principles and Practice of Obstetrics, De lee
1913.

ation of life.

To understand the fetal ballet of intrauterine life, a place to start is the shape of the uterus itself. The uterus is neither globular nor cylindrical but bean-shaped. The bulge is created by the lower back as it bends forward with pregnancy. This process is called lordosis. The back can bend as much as 5cm forward. Lordosis creates a central hump in the uterus which helps stabilize the fetus from excessive motion. It may serve as an obstacle to fetal rolling. Finally, this shape may guide the fetus to a head-down launch position by 37 weeks. (Figures 31,32,33)

Throughout pregnancy, this choreographed, instinctive behavior is directly linked to fetal survival. The fetus must develop intrauterine reflexes to cope with maternal movements and positions. Without these abilities, the fetus will fail in its journey to birth. A new perspective on the fetus which is at odds with the old perspective has emerged in the last 30 years. The fetus is now believed to be independent of the mother relative to its behavior. The fetus is also believed to control the onset of labor through its own local biochemistry such as its endocrine and paracrine systems.

These scientific facts put forth a view that the fetus is like a tadpole in a pond. Amphibian eggs are laid and left to fend for themselves. The tadpole develops independent of the mother through multiple stages. The human fetus develops in the pond called the uterus without any direct maternal care. It must fend for itself. Thus, the fetus is an active participant in its development and environment, and it decides when it is born. The fetus reaches milestones in the uterus just as a newborn reaches milestones of rolling, crawling, and walking over time. This scientific philosophy of fetal life points to behavior mechanisms of the fetus which would allow instinctive protection of the supply line.

Evolutionary changes had to favor a combination of relative relationships which would allow success of a reproductive assembly such as ours. Mammalian species all have cord-related complications. Monkeys, horses, and cattle have all been observed with cord problems. One difference is that these animals must be prepared to fend for themselves and ambulate at birth. The human is dependent at birth but is somewhat independent in the uterus.

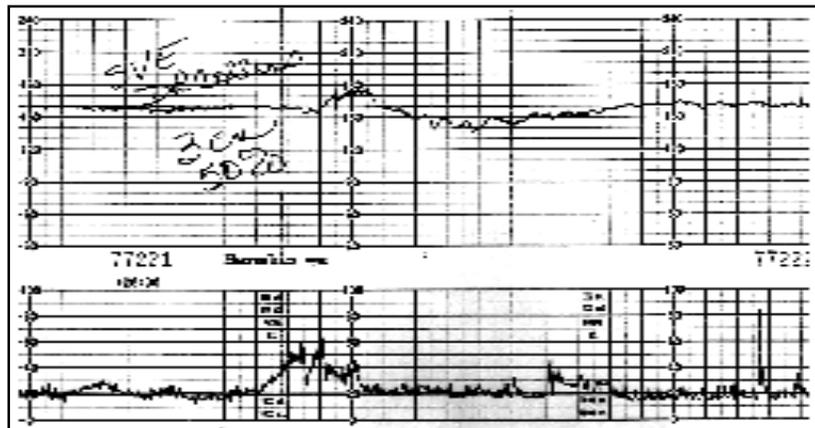
Cord compression can occur in the uterus with multiple siblings. This compression has been observed in pregnant rats. Experiments to reproduce cord compression have observed fetal jerking movements which are believed to relieve the compression. These movements have also been observed in human fetuses. It is believed that human single fetuses respond to cord compression with these reflexes.

A type of fetal jerking movement has been described as a hiccup. These movements are rhythmical and last for 10 to 20 minutes. Dr. Joseph B. DeLee, Professor of Obstetrics, Northwest-

ern University Medical School, wrote in his 1913 Principles and Practice of Obstetrics that hiccups were “one of the most interesting phenomena of intra-uterine life.” The movements are short, quick jerks of the shoulders and trunk. In connection with these observations, he wrote that “it was impossible to be sure that the infant was not suffering from asphyxia and was gasping for breath.”

A later observer of this behavior was researcher John Patrick, M.D., who studied this behavior with ultrasound. He believed that four 15minute hiccup episodes in 24 hours were normal. However, other insights suggest that this pattern should diminish toward term (37 weeks). A recent observation by Osamu Kurauchi, M.D. of Nagoya Japan School of Medicine supports the idea that startling, jumping, and writhing movements should diminish as the fetus matures. Studying “anenaphalic fetuses” (without brains), he observed that as the fully formed brain matures, these activities are modified and the behavior lessened toward term. In 1750, Dr. Smellie observed an infant with cord entanglement experiencing hiccups during vaginal delivery and afterward. We have reported that hiccups after 3 to 4 episodes in a 24-hour period may not be physiologic and that more than 3 to 4 hiccup episodes occurring during maternal activity may suggest cord compression. (Figure 34)

The nerve impulses that may cause hiccups may originate by way of the umbilical ring (bellie button). “Cholinergic” and “adrenergic” nerve terminals, or end nests, have been identified in the umbilical cord 20 cm out and concentrated toward the umbilical ring. These nerve fibers run with the umbilical



(Figure 34) Umbilical cord compression during uterine contraction showing fetal heart rate changes.

cal vein and sacral plexus to join the phrenic ganglion and celiac ganglion in proximity to the ductus venosus. The ductus venosus controls blood flow into the liver from the umbilical vein. It contains nerves and monitors fetal physiology. These fibers run with the vagal trunks and eventually may interact with phrenic nerve by way of the medulla and respiratory center. The phrenic nerve sends fibers to the pericardium, phrenic ganglion, sympathetic plexus, and hepatic plexus. Compression or stretch of the umbilical cord may lead to spasm of the ductus venosus and contractions of the diaphragm similar to a reflex. This activity may be originating at the umbilical ring which may be a pressure/blood flow sensor. These nerve pathways are basic structures of the

human body and how it senses basic body functions. Fetal behavior is directly linked to its physiology. The way it conserves oxygen, glucose, water and its reaction to these altered elements are remarkable adaptations of an interactive life as compared to a more dependent development.

The fetus is constantly faced with a changing physical compartment called the uterus. As the fetus grows and expands it first encounters a cavity which is twice its size. Between implantation and 12 weeks, the fetus (embryo) remains on a short tether, thick and restrictive to motion, it also repositions and reacts to maternal activity. The fetus therefore is stable relative to its position. This is not the case after 12 weeks. As the uterus grows and the cavity becomes several times larger than the fetus, the fetus now has “wings,” extended arms and legs which are moveable. Maternal activity can now distort fetal stability, and the fetus must respond to it or risk its supply line. Imagine an astronaut in space working on the space shuttle and spinning out of control. The line connecting life support to the shuttle will eventually fail.

In intrauterine space, as amniotic fluid expands the cavity, the fetus must develop a means to orient itself to react to maternal movements. This is accomplished by growth; fetal extremities resisting spin, roll, tumble and fetal reflexes adjusting fetal position.

As the fetus matches uterine growth between 20 weeks and 32 weeks, fetal reflex responses and mass act like a disc “brake” to “grab” the intrauterine anatomy to stay put. The fetus can “exercise” and develop, but it is able to remain “stationary” and not reposition itself (similar to working a treadmill). There is a difference between fetal movements and fetal motion (repositioning). The fetus is designed not to roll, spin, or tumble because these motions are deadly.

Fetal physiology as it applies to behavior of the fetus is primarily working to prepare the fetus for birth. It must have the ability to respond to changes that affect its heart rate, metabolic rate, neurologic status and position. These alterations are the source of fetal surveillance on which obstetricians depend to determine whether the fetus is compromised.

Fetal Senses

The main fetal senses are vision, hearing, touch, taste, and smell. Vision while developing is limited in utero. Eyelids are closed up until 26 weeks. Whether the fetus perceives light is unclear. At birth, newborns perceive shadows but do not clearly distinguish shapes. Vision would appear to be neither a primary mechanism for fetal behavior nor a means for the fetus to determine its position and status. In adults, vision is very important for day/night signals which manufacture melatonin and influence circadian rhythms. Fetal rhythms may be determined by maternal behavior and when meals are eaten.

Smell is not well developed at birth, nor is **taste**, which depends on smell. Nose plugs exist during fetal life; therefore, smell would not determine how a fetus presents itself. Chemical markers are important to many animal species for identifying food, dangers, and each other. Yet for human fetuses, smell does not appear to play a major role in the uterus.

Fetuses do not appear to identify parents by sight or smell at first. There is no need to forage for food in the uterus. Smell and taste only appear necessary in extrauterine life.

Swallowing, however, is connected to intrauterine and extrauterine life. The fetus must swallow fluid to make urine and develop the gastrointestinal tract. Suggestions indicate that what the mother ingests may reach these fetal senses through the blood stream. This “back door” chemosensory smell effect could play a role in how or whether the fetus ingests amniotic fluid. What does amniotic fluid taste like to the fetus? Is the taste covered up by the chemosensory effects, like putting sauce on a steak? These answers are unknown; yet whatever the purpose, it is probable that these senses do not have the same role in intrauterine life. They may play a role in initiating labor by responding to meconium discharged into the amniotic fluid. Meconium is the product of the fetal intestines which turns amniotic fluid green when expelled. Because meconium contains bile acids and other chemicals, ingestion may stimulate nerve receptors in the fetal nose and throat. These fetal nerve endings may contribute to neurologic signals reaching the hypothalamus, initiating the onset of labor.

Touch is an important sense for the fetus. Our knowledge of fetal touch is understood by what we know about newborns. The rooting reflex appears to develop after birth. Before birth, this sense appears to work in the opposite way. For example, a fetus seen on ultrasound turns away when touched on the cheek. Sucking fingers, toes and umbilical cords have also been observed on ultrasound. Yet turning away may be protective. Cord sucking may cause cord compression. In the uterus, turning away from objects may be beneficial. Touching and feeling the uterus, placenta, umbilical cord, and bony pelvis must be a part of fetal development. It may be a means for the fetus to locate comfort zones. Yet this may be more a function of pressure sensation than of feel sensation.

It is believed that the fetus can feel — at different degrees — from head to toe. The head is more “sense mature” than the lower body and extremities. The fetus probably prefers to feel itself and spends a lot of time touching with its hands. Fetal grasping is reflex-oriented. It is unknown how strong fetal grasp is (how much pressure it exerts). Can the fetus squeeze hard enough with its hand to occlude its umbilical cord with a pressure of 60mmHg or greater? It is also unknown whether touch or pressure influences fetal position.

Hearing and position (vestibular) sense are related. Another parallel sense is called “kines-

thetic sense,” or “where are my body parts?” These neurologic developments are the result of millions of years of evolution. The oldest sense is vestibular (inner ear). Even before the appearance of man, fish have had a position sense. It is not known whether the fetus senses gravity, weight and orientation.

The nerves to the ear mature around 24 to 26 weeks. At this point, the fetus can hear low frequency noises. It recognizes the maternal voice at birth. It may also have a sense of gravity (up/down).

What role does hearing play in the uterus? Although the answer is unknown, we do know that the fetus can be “startled” with sounds in the uterus. These “startle reflexes” may play a role in controlling fetal position. Known startle reflexes such as the Morrow Reflex are usually described after birth. This pattern of fetal movement is well known and can be seen by shaking a newborn. The arms take on a “hug” position with the hands going out away from the body, then inward.

What does this reflex look like inside the uterus? Imagine it as a fetal brake. The mother moves suddenly, and the fetus reaches outward to create contact between the uterus and itself. This has been observed on ultrasound, with the mother standing and lying.

There are other startle reflex patterns and fetal movements called “Tonic Neck Reflex” which extend the fetal arm, the opposite leg, and the head turns toward the extended arm. These reflexes are not intentional fetal movements as the fetus does not have coordinated movements. However, these movements do have an effect on helping the fetus change position.

The vestibular mechanism may be connected to the startle reflexes to position the fetus and provide a means of avoiding cord entanglement. Neural systems connected to the kidneys and related to the mid-section play a role in posturing independent of the spinal cord input. These extravestibular gravity receptors may also be connected to the cardiovascular system through a major nerve called the “vagus” nerve. This connection is very important to fetal stability. Auditory senses may assist in neurologic development for birth and help provide parent recognition.

How would these senses work to protect the fetus when it is active and asleep? When the fetus moves, it does so by movement of the midsection and the neck. These muscle groups are stronger than the extremities. It is often observed that the fetus “kicks” and “jumps” in utero. These activities are not coordinated and are not deliberately initiated by the fetus.

When the fetus repositions itself — as opposed to movements — different factors are involved. For example, the fetus may sense inner ear pressure, which may change with position; the fetus may sense contours and prefer the left lateral position. In addition, auditory awareness of the placental souffle or maternal heart beat may predispose the fetus to the head down/left lateral posi-

tion. When “uncomfortable,” the fetus may attempt to roll, tumble or somersault, using its startle reflexes to find “comforts.” This becomes more difficult after 32 weeks as the fetus is now larger than the uterine cavity and must fold itself into a “fetal posture.” If in the process of repositioning it becomes cord entangled, it may sense this through resistance to movement or the consequences of disturbed blood flow, oxygenation, and nutrient delivery.

Fetal Physiology as it Relates to Cord Compression

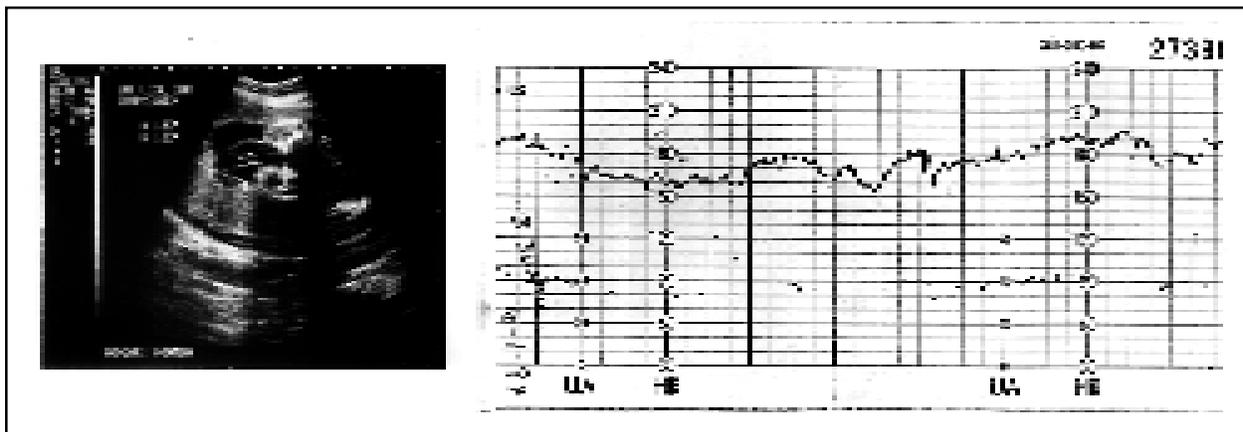
When the umbilical cord is compressed, the fetus immediately senses it. If the compression persists, the fetus will begin to undergo heart rate changes. These changes are sensed chemically (chemoreceptors) and physically (baroreceptors).

The first response depends on how much of and how long the cord is compressed. Complete compression causes the fetus to notice changes in blood flow and blood pressure, oxygen decrease and carbon dioxide accumulation. The heart slows and the blood pressure rises. Eventually this changes to a decreasing heart rate and decreasing blood pressure. Chemical signals are released to modify this response until it is corrected.

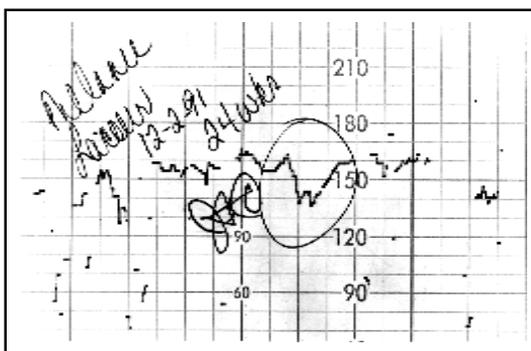
Usually a one-time, one minute, 100% compression of the cord takes 5 minutes to completely correct. But within that one minute, oxygen levels have decreased 50%, and the fetus must reset the valuable energy and chemistry it has expended. In a recent experiment, complete cord compression for 5 minutes required 30 minutes for recovery. Continued 5 minute compressions every 30 minutes caused fetal decompensation. This happens because the fetus cannot reset all of its hormonal, chemical and nutrient baselines quickly. Some refueling takes longer to rebuild than others.

The main fuel of the fetus is glucose. The fetus has its own store of crystalline glucose called glycogen in the liver, heart muscle and elsewhere, but it does not use this emergency supply unless it is completely deprived of maternal glucose. This is important for its survival and proper cardiac function if stressed. When the fetus senses blood flow interruption, it also senses a variety of organ reactions. The liver reacts by changing blood flow to a vessel called the “ductous venosus.” This vessel also directly connects the fetal heart to umbilical blood flow providing the fetus with more oxygen that is already available. The fetus shifts blood from its extremities to favor enough oxygen to its heart and brain. If the blood flow derangement persists after one minute, other systems join in to adjust for the loss of oxygen and nutrients. In addition to liver blood flow changes, the fetus experiences blood shifts away from its intestinal blood supply and kidney blood supply. Renal (kidney) blood flow is altered which, if sustained, will begin to change renal function. Adrenal glands responsible for releasing stress hormones now secrete cortisol, a stress steroid,

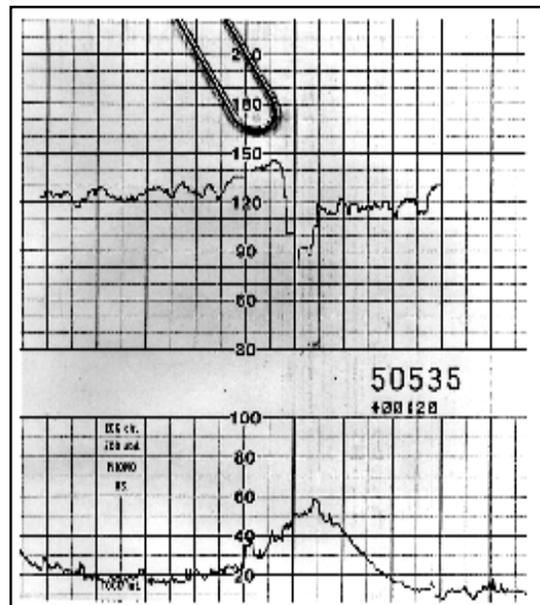
and immediate-acting adrenaline called catecholamines. These chemicals alter the fetus' cardiovascular system (heart rate, blood pressure, pulse) and stimulate the fetus to adjust its position if related to cord compression. This positional change would be invoked by the fetus bending its thorax and neck and by inducing hiccups. All of the fetal reflexes are now initiated and modified to achieve normal state. Once these events take place, the fetus is probably tired, like an athlete running a sprint, and it needs to recuperate. As the fetus rests, its urine changes composition and becomes more diluted, releasing $\text{Na}^+\text{K}^+\text{Cl}^-$ ions. Similar events probably take place in the fetal intestine; however, if the fetus used liver and heart glycogen, it cannot be replaced. If the glycogen store is low, it could create difficulty for the fetus if it continues to be stressed and is unable to depend on its only fuel storage. Heart dysfunction could eventually take place. (Figures 35-37)



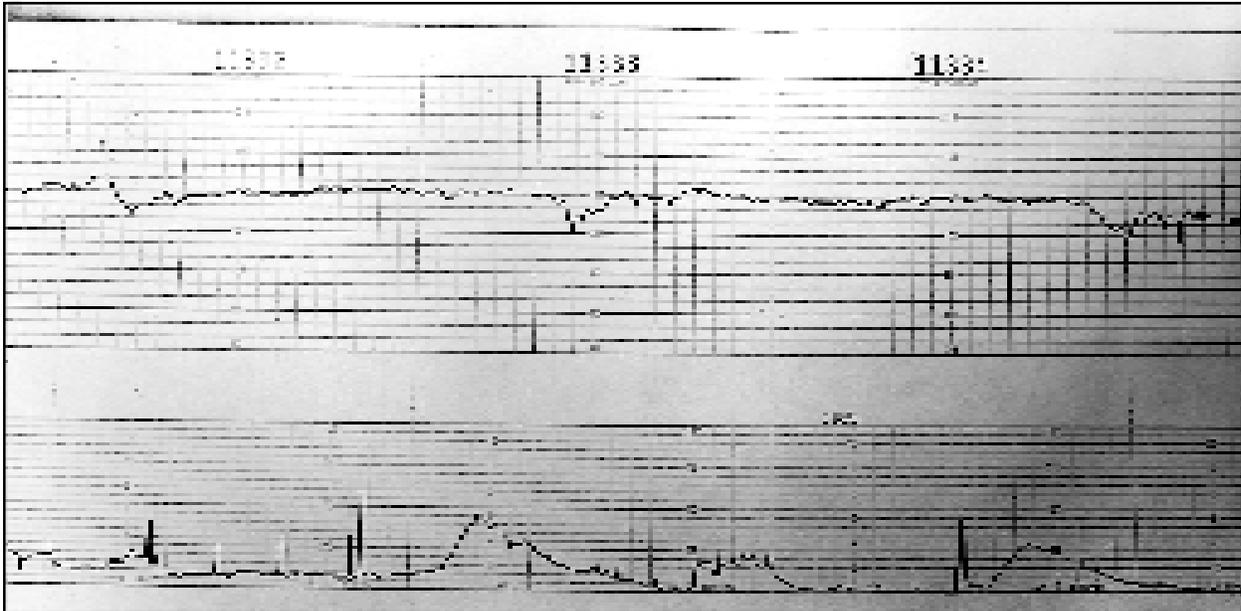
(Figure 35) Cord compression leading to an increase in heart rate.



(Figure 36) Cord compression leading to a "W" sign.



(Figure 37) Cord compression leading to a "Lambda" sign.



(Figure 38) Cord compression during labor leading to fetal heart rate decelerations with an increase in baseline.

Fetal Behavior and Physiology Secondary to Cord Compression

In animal models, usually fetal sheep, repeated complete blockage of the umbilical cord changes the way the kidney excretes basic electrolytes. These molecules are important for basic cell function $K^+Na^+Cl^-$ ions. If the cells of the body are unbalanced, they do not work properly. Improper cell functioning is similar to having a bad stomach virus with diarrhea and vomiting. In addition, this change is connected to the secretion of a hormone called vasopressin. Vasopressin is responsible for moderating blood pressure. When the fetus is stressed it attempts to change its function until it relieves the stress.

If cord compression is not relieved, further physiologic changes begin, the purpose of which is to conserve oxygen. Blood flow can be taken from the extremities (arms and legs), other organs (such as the lung) and preferably shunted to the brain and heart. This preserves the brain and heart at the expense of the body and gives the fetus time (10 to 15 minutes) to solve the problem of obstructed umbilical blood flow.

If the fetus cannot correct the cord blockage, final changes begin to unfold. The fetus can tolerate these conditions as long as it has oxygen and glucose and it is not losing blood. But beyond 15 minutes, the fetus begins to decompensate and experience tissue damage. The most vulnerable tissue is nervous tissue. As the brain loses oxygen, it turns to a back-up nutrient called lactic acid. The problem with this brain food is that it does not metabolize cleanly and leaves buildup mol-

ecules called carbon dioxide; this situation causes the brain to swell, prompting blood vessels to close. Brain damage then occurs. The degree of damage depends on many factors such as the age of the fetus at the time and the degree of oxygen loss to the tissues. As the lack of oxygen increases, the heart begins to fail. Not only does it malfunction in its role as a pump, it also begins to “short circuit” and develop arrhythmia, which is irregular heart beats. These electrical conduction defects put the fetus at risk for heart stoppage. If the fetus is low on oxygen (hypoxic), high on lactic acid and carbon dioxide (acidotic), and experiences heart arrhythmias, it will die.

The fetus has behavioral patterns that can create cord compression and release. These reflex movements and organ adaptations have evolved over millions of years. The overall relationship is a vigorous fetus having the ability to develop in the uterus without injuring itself, its cord, or its placenta while growing. The process of labor alters all these behaviors and recruits a new set of protective mechanisms designed to evade disruption of the supply line. During labor, compression patterns can change from a single short episode to repetitive cord compressions to prolonged compression where complete blockage forces immediate delivery. A typical example of complete blockage is a prolapsed cord where the cord enters the vagina before the fetal head. The cord can be compressed between the bony fetal head and bony maternal pelvis. Usually fetal compromise will occur if delivery does not take place in 10 minutes.

A more detrimental accident is blood loss. Placental separation due to a short cord, umbilical cord rupture due to a defective cord, or a ruptured umbilical vein hematoma prompts not only blood loss but a lack of oxygen as well. These types of accidents can cause fetal death in minutes. Little can be done under these circumstances as these infants need immediate, rapid treatment with blood, fluids, oxygen, and support. If they survive, it is usually with damage.

The animal model which is not familiar to most is the one which simulates intrauterine, prelabor, intermittent partial cord compression. This will be discussed in the next chapter.

“Management and treatment options for umbilical cord complications will follow an understanding of the basic pathophysiologic mechanisms.”

Leon Mann, M.D.
Indianapolis, Indiana
1986

“We rely greatly on information from animals; too much since there are great variations between species—we know so little; measurements of flow of the umbilical (cord)—are imprecise, and we have few measurements of human fetal arterial blood pressure, or of its changes with age—. Hence, much is speculative!”

G.S. Dawes, D.M. FRCOG

Fetal Physiologist

Oxford, England

1995

Chapter 4

What Animal Research Teaches Us About Umbilical Cord Accidents

“Animals will continue to be as vital as the scientists who study them in the battle to eliminate pain, suffering, and disease from our lives.”

Heloisa Sabin

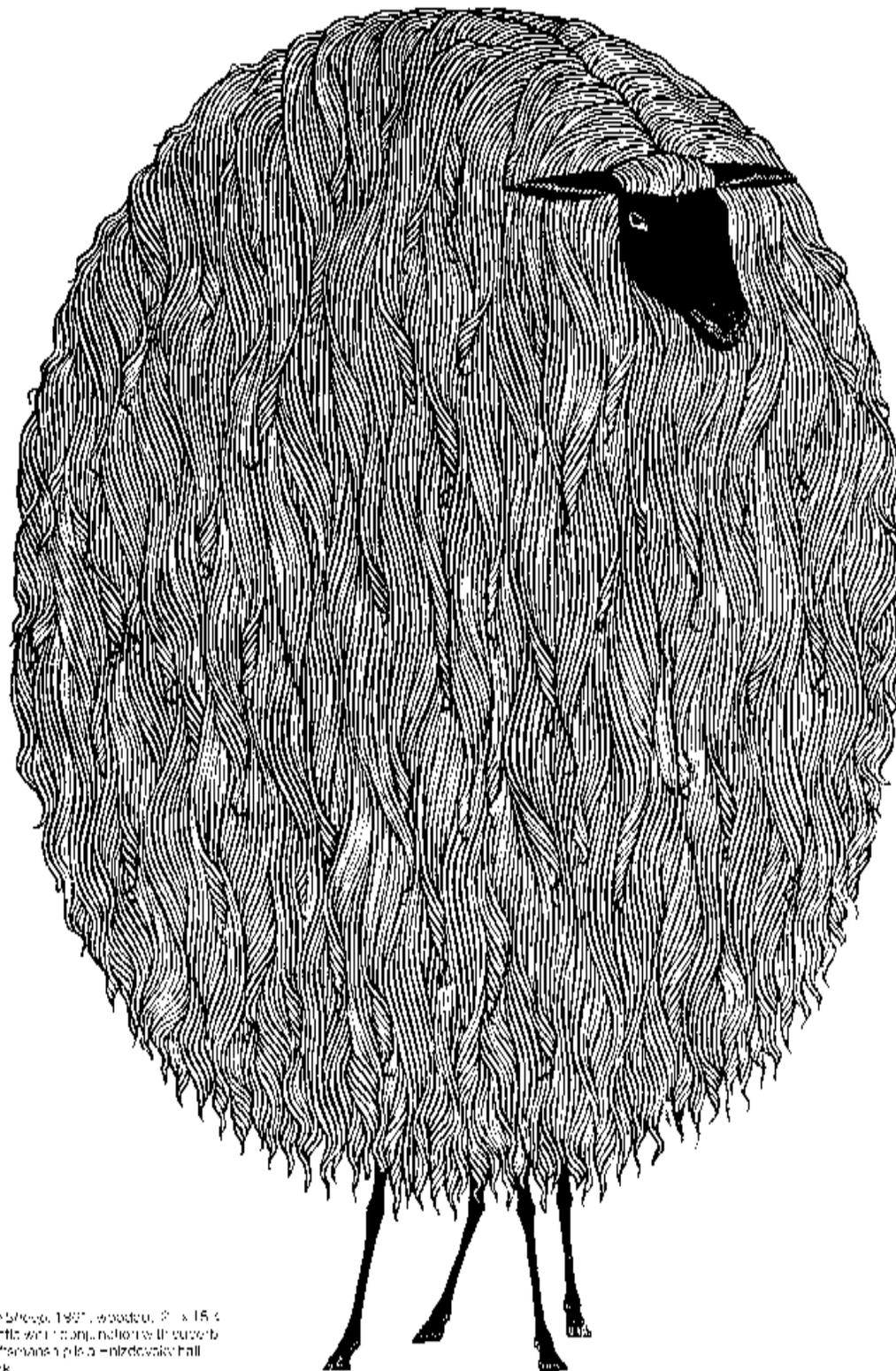
Speaking about Albert B. Sabin and Polio Vaccine research, “Animal Research Saves Human Lives,” Wall Street Journal, October 1995.

Some of our knowledge about how the fetus behaves and is affected by supply line disturbances comes from animal research. The ability to solve the problem of umbilical cord accidents may depend on future efforts involving research in animal models. From mice to rats to dogs to sheep to primates, valuable insights may only be possible using these heroes of research.

Numerous dedicated world class researchers depend upon animal models for their work. Many have contributed invaluable insights into how the fetus works. Translating animal model research results to humans is not perfect and at times very different. Sometimes, too, human research gives insights to help animals.

An important animal model for fetal physiologic research is the fetal sheep. This works out fairly well due to sheep tolerance of manipulation and similarity of human biology. Through sheep, scientists study fetal reaction to various forms of oxygen deprivation, one of the more poorly understood areas of obstetrics. Fetal sheep can be chronically oxygen depleted or acutely oxygen deprived, and the response to each situation differs. The time in gestational weeks and duration of a lack of oxygen in these models can be varied to show different fetal responses.

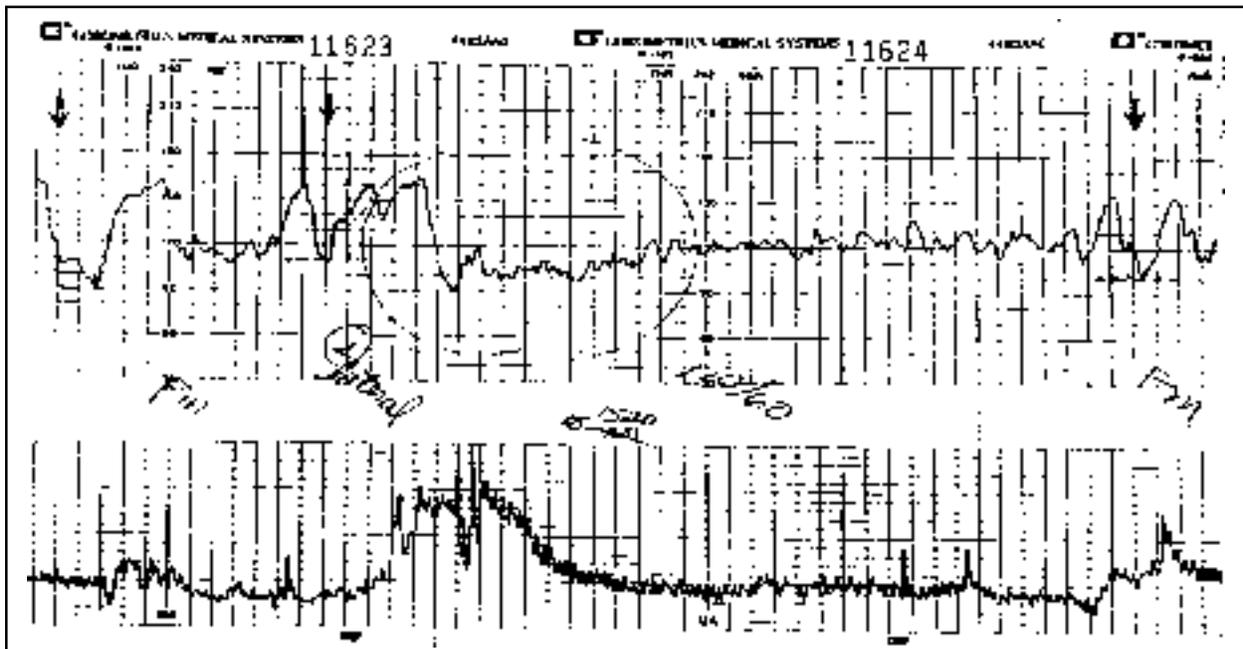
There are several ways to study these events. One is to limit oxygen to the mother of the fetal sheep while the mother is under anesthesia. This, in turn, limits oxygen to the fetus. Another method is to limit blood flow to the uterus. Special techniques exist to reduce placental blood flow and simulate “intrauterine growth retardation,” which is thought to be due to similar conditions. Direct blockage of the umbilical cord can limit blood flow and oxygen delivery in varying degrees. These studies help scientists understand all the ways a fetus can be compromised.



John Shove, 1907, woodcut, 21 x 15 cm
Gentle with a man, in line with the picture
can't be seen a picture - in the dark hall
meek

A specific animal model in sheep fetuses blocks the umbilical cord to simulate umbilical cord accidents. Several such studies have suggested insights into how supply line interruption affects the heart, brain, kidneys, and metabolism of fetal sheep. When the umbilical cord is compressed in these models, characteristic fetal heart rate changes take place. One pattern type is called “variable decelerations.”

“Variable fetal heart rate decelerations” are changes in heart beat-to-beat patterns which cause the heart rate to decline below normal levels (110 beats per minute) (Figure 39). This is also a clinical sign used to determine whether a fetus needs immediate delivery. To understand how this works, fetal lambs had “occluders” placed around their umbilical cords while in the uterus. The occluders were opened and closed by timed pumps to simulate possible intrauterine events. Highly specialized, sophisticated equipment was used to measure the oxygen, acidity (pH), and other chemistries of fetal physiology.



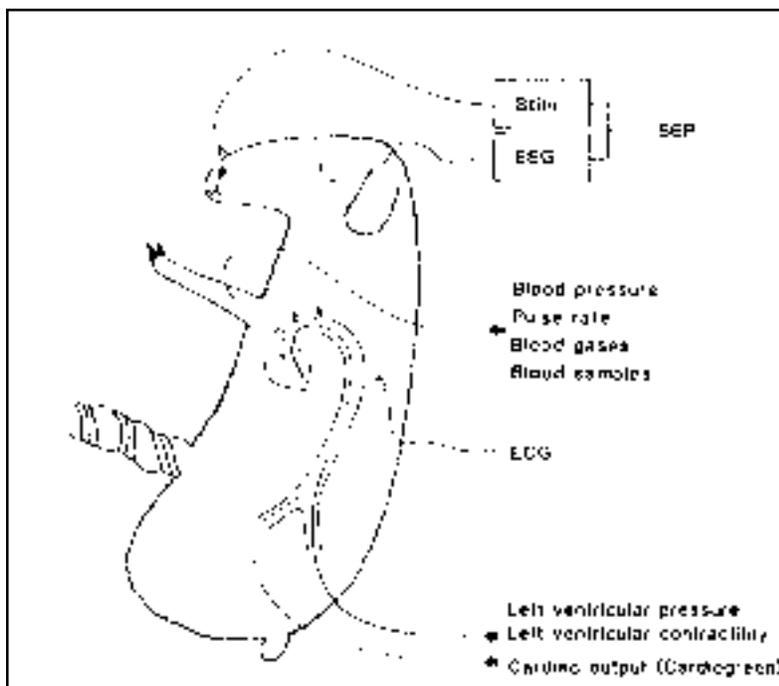
(Figure 39) 110 beats per minute

These studies showed fetal stress within the first 30 minutes of a two-hour experiment. The fetal heart rate reacted immediately, and the body chemistry changed quickly. Characteristic heart rate patterns called W signs, U signs, and V spike signs appeared. Fetal stress hormones began to rise and eventually became depressed or modified. In the sheep fetus, this suggests that time is an important factor when umbilical cord compression is occurring.

Considering this on a human level, correction of cord compression over a reasonable amount of time is important. If the human fetal heart works like the sheep fetal heart, then neurological

regulation of the fetus deteriorates with progressing “accedemia” and “hypoxia” caused by these compressions. Even if fetal death does not occur, the effects on the heart may be enough to permanently change the heart muscle. For example, a drop in fetal blood pressure creates a decrease in coronary blood flow and O₂ delivery directly to the muscle. Indirectly, chemical products from the placenta abruptly enter the heart after release of cord compression. These, in turn, can affect the heart muscle with a modified oxygen called “oxyen free radicals.” This excited molecule can burn heart muscle and change it. Vitamins such as C&E are called free radical quenchers. They can react with the excited oxygen molecule and return it to a normal state.

The chronically instrumented pregnant sheep and fetus have been used to study the effects of umbilical cord compression on neurologic function (Figure 40). An overall view of complete cord compression showed an increasing tendency of the fetus to conserve blood flow between the



(Figure 40) A schematic presentation of the fetal preparation.

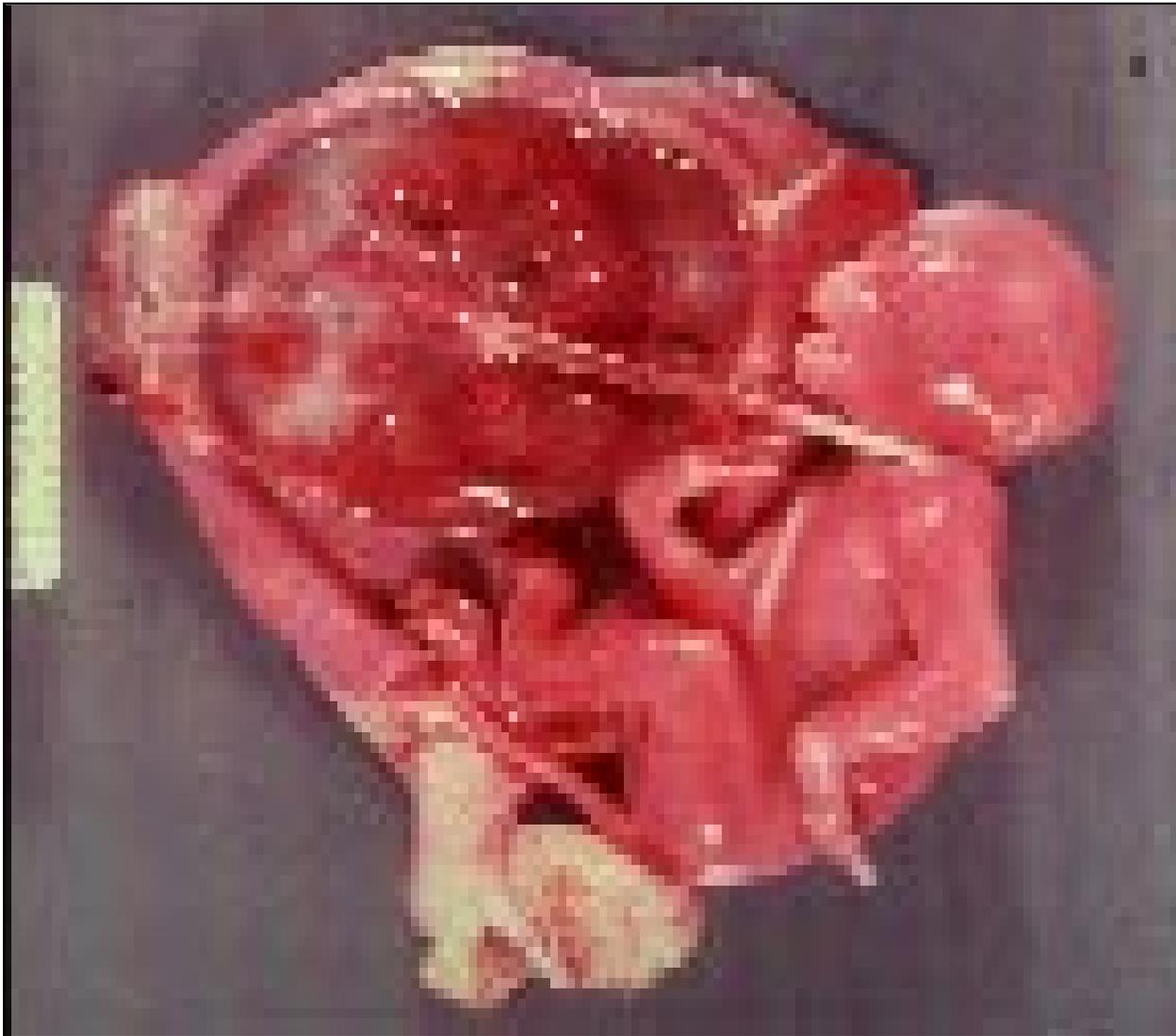
heart and the brain using oxygen from the limbs. This, in turn, directs oxygen to the more fragile neurologic tissue and away from the more durable gastrointestinal tract, muscle, and skeleton. This diversion appears to protect the brain and heart in that it buys time for a correction (see Chapter 3).

These experiments give scientists a chance to look at the brain's reaction to cord compression. For example, depending on time, duration and degree of blood flow inter-

ruption, various changes are seen. One change is edema (swelling) of the brain tissue. If severe, edema can be damaging. It is known that normal infants with uncomplicated births have edema for several days. How all of this affects the fetus at different ages is unknown since animal models in mature sheep suggest that umbilical cord compression can cause varying types of damage with different insults of oxygen deprivation.

Complete cord compression with complete loss of blood flow and oxygen may have several effects in term infants. In term sheep these events cause hippocampal (learning center) damage after 10 to 15 minutes. Striatum (neuronal) damage occurs after repeated 5-minute compressions.

This area contains white matter and is located lateral to the ventricles of the inner brain. Important cross connections to the brain's right and left hemispheres are located in the striatum. In different experiments, 50% blood flow interruption caused brain swelling, heart rate changes, and endocrine changes. Brain edema can press small blood vessels on the underside of the brain against the ridges of the skull and selectively reduce blood flow to specific areas. Heart rate changes can lead to arrhythmia of the heart and heart stoppage. When the cord is clamped and released over time in term sheep, other patterns of damage occur in the (white) brain matter. Similar patterns have also been observed in primates. In addition to neurologic changes, blood flow shifts from fetal organs and extremities to the placenta and heart are also documented (Figure 41). These patterns are important to define if the mysteries of cerebral palsy and learning deficits are to be understood. During the 36th to 40th week of human gestation, significant damage appears to be possible due to combina-



(Figure 41) Nuchal cord stillbirth in a primate fetus.

tions of hypotension, hypoxia, and stress caused by prolonged cord compression.

While the fetal heart and brain are being protected during cord compression, the fetal adrenal gland is also being protected. The adrenal gland sits atop the kidney and provides stress hormones and chemicals. The adrenal gland usually reacts to danger in a “fight or flight” manner. For example, if stressed or compromised, the adrenal gland can “turn on” the “launch signal” prior to its due date. In the sheep fetus, when cord compression reduces blood flow, extremity (limb) circulation is shunted in part to the adrenal gland. This enhanced blood flow allows chemistry from the gland to be excreted to the fetus.

The gland has two parts: an inside core and an outside capsule. The immediate response is to produce “adrenalin” (norepinephrine, epinephrine). These chemicals raise fetal blood pressure and stimulate the fetal heart to beat faster. In fetal rats, it may cause the fetus to “jerk” or move. While this occurs, the adrenal gland produces a stress hormone called cortisol which protects the fetus from “collapsing” or having a “nervous breakdown.” These chemical signals go to the brain of the fetus which, in turn, sends signals to the mother which may ultimately cause release of oxytocin and perhaps vasopressin, all of which may initiate contractions.

The liver is another organ which may be stimulated during umbilical cord compression. An important fuel for fetal life which is stored in the liver is glucose. This is in crystalline form and present in muscle and other tissues. One study examined glucose metabolism in fetal sheep and noticed increased glucose hormones (glucagon and insulin) during decreased O₂ exposure. The placenta, cord, and muscle contain glycogen which, once used, is not restored. In chick and mouse fetal heart samples, the distribution of glycogen inside the cell changes with time. With maturity glycogen increases in the cell structure called the mitochondria. This is the cell “engine” and provides energy for cell mechanisms. Muscle cell glycogen stays about the same over time. The overall glycogen in the cell decreases from 1 day to 30 as if concentrated in the mitochondria. Only when the fetus turns to a post-delivery metabolism can it rebuild glycogen. This change in stored fuel may be important for other fetal conditions.

In addition, vessels in the liver tend to direct blood flow through an important vessel in one of several “fetal shunts” which are not needed after birth. Blood flow shunts are special vessels which redirect blood flow during fetal life. (Once born, these vessel “detours” are permanently closed to allow adult life.) Disturbances in these shunts may play a role in sending signals to the adrenal gland during stress through the right phrenic nerve. This nerve branch, which matures by 24 weeks gestational age, could be an important “emergency alarm” that plays a role in quickly turning on stress chemicals when necessary. Umbilical cord compression also has direct effects on

these shunts and their ability to “stream” blood to the heart.

Lungs are affected by these changes of blood flow through shunts in sheep animal models. Cord compression of 50% reduced lung interstitial blood flow. By altering O₂ delivery, different patterns of damage are established. One of the effects of cord compression on lungs is the associate development of persistent pulmonary hypertension. This pathology has been demonstrated in a sheep model. A recent computer simulation model found that as the fetus shunts blood from the lower body to protect the brain and heart, lung oxygenation falls acutely. All current published facts point to a relationship between altered umbilical blood flow and lung maldevelopment.

Other lung effects unaddressed are fluid balance and surfactant production. The lungs are bathed in amniotic fluid which participates in lung alveolar (air sacs) growth. It is unknown whether disturbances in “Water Management” can change the lungs ability to mature. If the forty different lungs cells are not receiving proper blood flow they may not be producing chemistry to support lung functions such as O₂/CO₂ exchange. If fluid management is disturbed too much or too little fluid may alter how the fetus will breath at birth. To date no studies exist which consider amniotic fluid/lung function and relationships with chronic cord compression.

Thanks to expert studies of animal models, fetal responses to umbilical cord compression are known. A basic understanding of vital organs such as the brain, heart, kidney, lung and adrenal gland will help determine how to help the stressed human fetus. The ability to study blood samples from both the mother and fetus will lead to methods to test the fetus and find the one needing delivery.

Some of the biochemical changes that can result from umbilical cord stimulation are as significant as the organ, blood flow, and neurological changes. As the fetus experiences more stress, the tissues of the umbilical cord can secrete chemistry to react to the changes taking place. The local tissue reactions may be premature changes that would occur only during labor. Because the tissues cannot individually tell the difference, many false starts can take place. Local reactions in the umbilical cord by molecules released from the cells that line the umbilical vessels can cause momentary constriction or closure of the vessels. At birth, these molecules permanently close those vessels to prevent blood loss.

These small dynamic systems are constantly in balance. Thousands of these systems probably exist, some more immediate and more important than others. These systems are sometimes called “autocrine” and “paracrine” modulators and regulate local (cellular) chemical reactions. An obstetrical interest is the potential for a remedy to maintain blood flow. Discovery of a modulator that can be infused to the mother would be beneficial when there is evidence of blood vessel con-

striction. Animal tissue studies can greatly enhance the understanding of these molecular effects.

Studies in both human tissue cultures and animal tissue cultures have broadened our knowledge of umbilical cord vessel “endothelial” cells. These are cells which “coat” the inside of umbilical arteries and veins. They produce the chemicals which respond to the conditions that occur moment to moment with the events of the placenta and fetal physiology.

LIST OF SOME PARACRINE/ENDOCRINE/AUTOCRINE MOLECULES

Oxygen - Several forms (modulators of other molecules)

Nitric Oxide	-	Relaxes placental vessels
Nitrite	-	Byproduct of oxygen and nitric oxide - inactive
Peroxynitrite	-	Byproduct of active oxygen, toxic to cells and active in the membrane of cells
Prostacyclin	-	Vasodilates placental vessels
Thromboxane	-	Constricts placental vessels and modulates coagulation
Endothelin-1	-	Constricts placental vessels
Epidermal Growth Factor	-	Tissue modulator
Prostanoids	-	Modulate coagulation of blood in umbilical vessels
Prostaglandins (PGE₂)	-	Constrict umbilical vessels at birth
EGF + TGF	-	Constrict and regulate cord function
Xanthine/Purines/ Adenosine	-	Vasodilate placental vessels
Catecholamines	-	Stress molecules which modulate vessels
Nicotine	-	Vasoconstrictive
Caffeine	-	Unknown
Chocolate	-	Unknown
Tea Herbal	-	Unknown
Anesthetics	-	Bupiracaine - constriction of umbilical vessels 2-chloroprecaine - dilation of umbilical vessels

It is known that the amounts of these “autocoids” differ from umbilical vein to umbilical artery and from the placenta to the fetus. Anticoagulation autocoids (blood thinners) are higher in the umbilical vein; vasoconstrictor autocoids are concentrated toward the fetus which means that they are important in stopping blood loss from the cord when it is severed.

Epidermal growth factor which makes “PGE²” is located less on the placental end than the fetal end. This difference allows the constriction of cord vessels to begin at the placental end and peristalsis (squeeze) blood toward the fetus. This trick of nature insures an adequate blood count for the fetus. If this fails in large animals such as cows and horses, the newborn calf or foal will die of shock due to lack of blood volume.

Numerous “regulators” also exist outside and inside the umbilical cord. Amniotic fluid contains molecules from fetal metabolism, and blood circulating through the cord has molecules which are important to blood flow. It is amazing that any of us are born in one piece. These multiple systems can remain in balance and can adapt to disturbances. What is unknown is the limit of that adaptation. When does failure begin, and how long after failure does damage begin? Sometimes damage is immediate, sometimes it is confined, and sometimes it is chronic. To know these answers is to discover important ways to protect the fetus at risk.

Animal models also allow research into other reproductive byproducts such as the placenta. Determining the effects of fetal derangement on placental function is most important. Where the fetus can avoid injury, the placenta may not. No matter how vigorous the fetus is, it is in danger when the placenta deteriorates. The differences in placental structure from animal to animal allow varying effects. Sheep have multiple placentids where monkeys have one placenta like humans. Chimpanzees are better examples of human placental design.

Morbidity

Other Effects

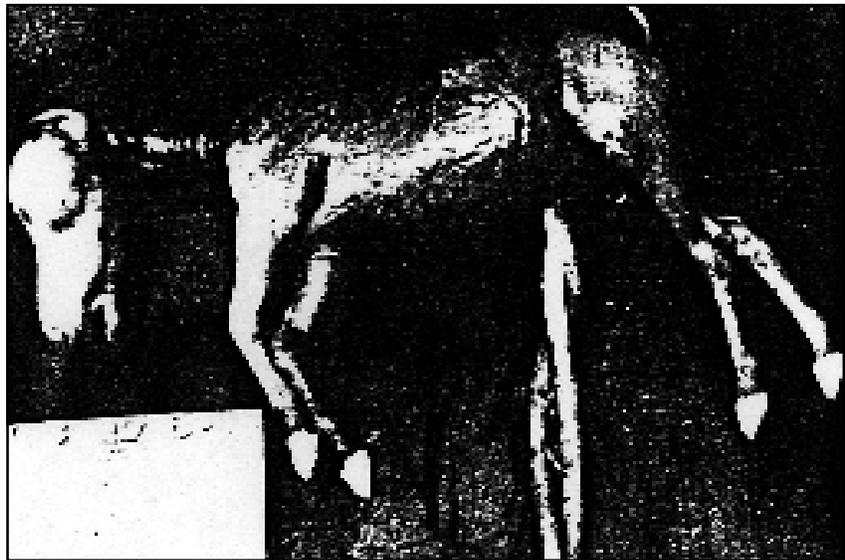
Placental changes are important to study separate and apart from fetal effects. These placental changes may or may not add to the fetus’ ability to tolerate intermittent umbilical cord compression. The placenta has large reserves similar to other organs, where loss of placental tissue may be comparable to a loss of hearing in one ear, sight in one eye, or function of a kidney. The fetus can function and survive, but it may not grow and develop to 100% of its capacity. Cord compression may be a stimulus to induce such a compromise.

It is theorized that if the umbilical blood flow is slowed, then placental blood flow is also slowed. When this happens, blood “thickens” in the small spaces in the placenta called the “vil-

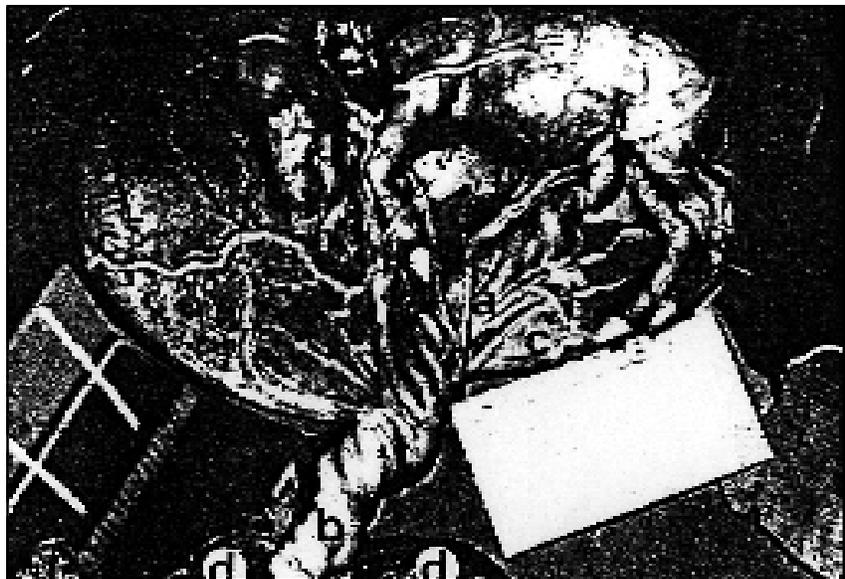
lous” vessels and “clots” like gelatin. This in turn causes “thromboses” of the “intervillous” space which contains blood flow from the mother. The result is devitalized placental tissue and loss of that placental nutritional space. If enough dead space is developed, the fetus can be compromised. Thus, an indirect effect of cord pathology may be the “secondary” injury of the placenta even if the fetus does not itself become primarily injured.

The umbilical cord complication observed which causes such a pattern is torsion (Figures 42 & 43). Animal placentas have been studied where torsion caused stillbirth of thoroughbreds (Figure 44). Thrombi were seen in these placentas along with thrombosed surface vessels. Human placentas have been observed in cases of torsion where surface vessels were similarly thrombosed. The finding suggests multiple small blood flow derangement episodes prior to death. Does this mean these events can be prenatally detected? It is possible that one day ultrasound or maternal blood sampling will detect these changes.

Another placental change which is more extensive than infarction of tissue is called chorangiosis. It is suggested that this change is due to low oxygen levels in the fetal circulation. This alteration of placental blood vessels looks like a “rash.” The vessels are dilated and



(Figure 42) Fetus from a 164 days gestation: no torsion or less than, 1 revolution clockwise.

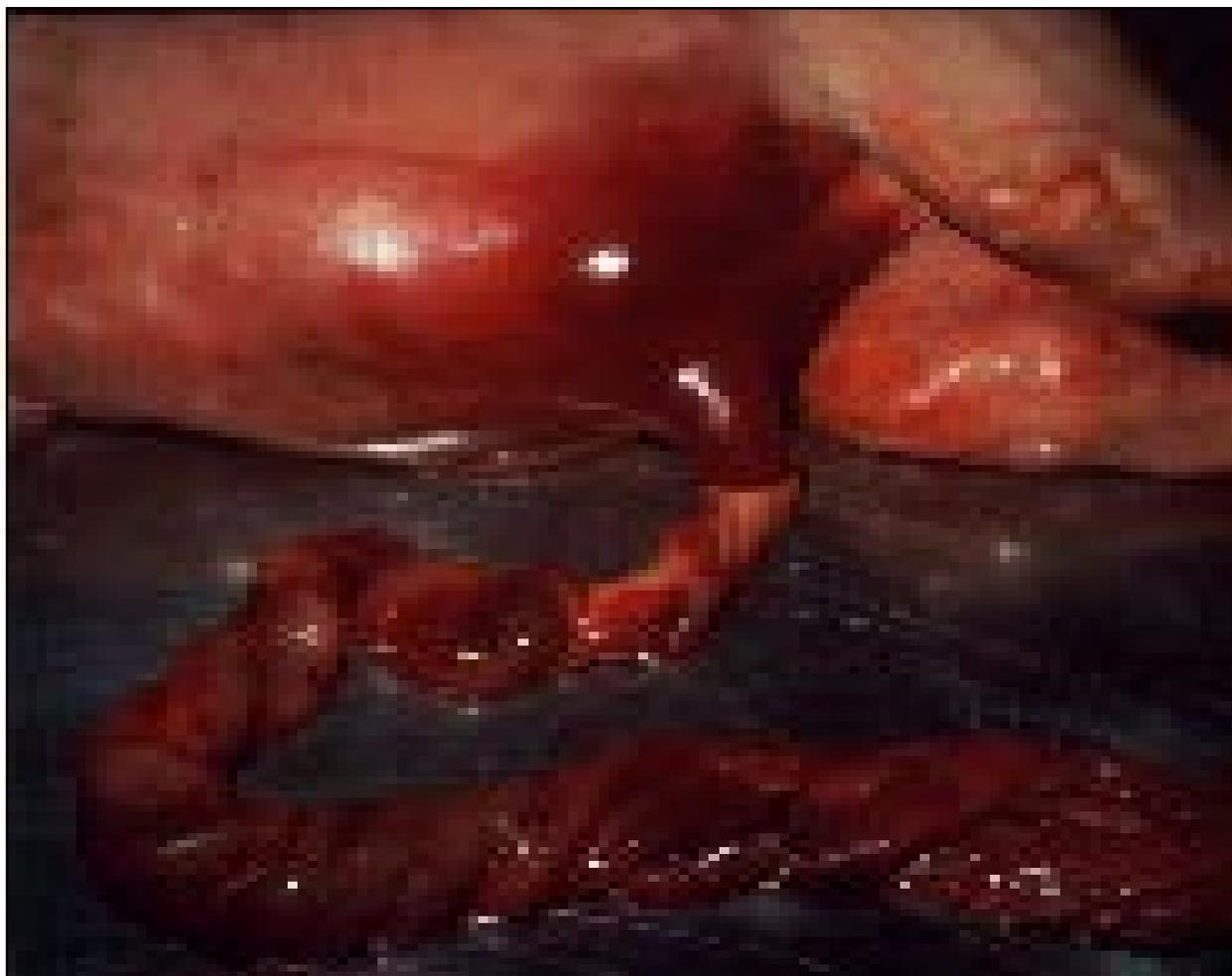


(Figure 43) A pregnancy of 134 days: five and four clockwise torsions of the amniotic (a) and the allantoic part (b) of the umbilical cord respectively. Closed allantoamnion (c).

full of blood cells. This change allows more surface area to grab more oxygen from the mother. How much change is tolerated before fetal harm occurs is not clear.

Chorangiomas and similar placenta changes have been studied in llamas and goats from high altitudes, especially in Chile. Because of low oxygen tensions at elevations above 10,000 feet, these animals provide an important insight into adaptations to tolerate chronic low oxygen states. These changes seem to suggest that the chronically hypoxic (low oxygen level) fetus compensates with blood flow and blood cell expansion. To see similar changes in a fetus with cord entanglement suggests similar principles are involved.

All in all, multiple questions must be answered to solve the mysteries of umbilical cord compression. The answers may be forthcoming in each method of animal modeling used. Because of each unique characteristic of each species, study of the whole assemblage will be necessary to define umbilical cord compression effects on the fetus.



(Figure 44) Torsion stillbirth in a fetal horse

Chapter 5:
**Management of Umbilical Cord
Anomalies, Abnormalities, and Complications**

“Fetal loss from umbilical cord abnormalities—a difficult case for prevention.”

**A. Ghosh, M.D.
Queen Mary Hospital
Hong Kong, 1984**

Managing the umbilical cord during pregnancy, labor, and delivery needs more discussion in obstetrical forums. In 1989 a publication from the U.S. Department of Health and Human Services entitled “Caring for Our Future in the Content of Prenatal Care,” describes specific pregnancy “conditions or hazards.” It comments that “structural abnormalities of the placenta are significant causes of poor pregnancy outcome.” No other specific mention of umbilical cord related events are listed and no specific screening or management is suggested. This report of the Public Health Services expert panel on the content of prenatal care did not address the issue of umbilical cord accidents but seemed to acknowledge an association with fetal harm.

Increased discussions of cord accident management are also important because this topic is virtually undeveloped and poorly defined. This fact is concerning because more infants are affected with cord complication than are with Polio, AIDS, SIDS or B-strep. The effects of umbilical cord related complications can be as dramatic as a term stillbirth or as subtle as learning disabilities. It is consistent in that fetuses are being lost to cord accidents on a daily basis.

The problem must not be understated. Once it was discovered that the polio virus caused harm, Dr. Salk was able to construct a management solution called polio vaccine. Umbilical cord accidents are causing stillbirth, asphyxia, emergency C-sections, fetal distress, and neurologic damage which may consist of learning disabilities and cerebral palsy.

How are these fetal harms to be prevented? Part of the difficulty in prevention seems to be in believing that an association exists. These relationships are difficult to demonstrate statistically. One analysis of cerebral palsy infants stated that looking at “cord prolapse, body loops, and [“long cords”], there were significant associations.”¹ (Figure 45).



(Figure 45) 20 week fetus with nuchal cord type A x 1.

All these insights are difficult to prove because the reviewed and relied upon information is incomplete and full of statistical pitfalls. What can be done at this moment in time is to discuss and make clear the potentials of umbilical cord accidents.

The attempt to manage cord problems during labor may go back to Zi-Ming Chen (A.D. 1237). His suggestion was to remove a shoulder loop by hand. “Doctor should ask the mother [to] lie on her back and be at rest, gently press its shoulder downward, and strip off the cord. Don’t order the mother to exert her strength till the fetus positions itself properly.”²

In 1741, British obstetrician Dr. John Burton described several cases of cord management where delivery was impeded by a short cord and an entangled cord.

“I was sent to a patient at Healey Manor who had been in labor about 30 hours.— I did imagine that the umbilical cord must be too short, and then I reached the string and found it fully stretched with the placenta strongly adhering to the womb. I therefore broke the string [cord] and delivered the woman...”

“I was sent to a person at Enwood.— I introduced my finger a little further and found the umbilical cord sat about the child’s neck. I therefore twisted that part of the cord, which reached from the child’s neck to the placenta, about my two forefingers and with my thumb broke it, withdrew my hand, when the child soon followed.”

Dr. Nelson Sackett in 1933 described a case where he reached inside the uterus, “encountered two loops around the baby’s neck” and extracted the infant in the back position (version extraction) rupturing the cord in the process.

A study reviewing over 1,000 births recently concluded: “From the point of avoidability of prenatal deaths, this study indicates that there is no clinical pattern and therefore no place of treatment for the obstetrician to save those babies which are killed by their own umbilical cord.”

Harold McLellan, M.D.

Australia, 1988

This conclusion was based on traditional obstetrical care. There was no attempt to use tools such as ultrasound and fetal monitoring as a preventative measure.

Some researchers have taken a different approach. In a case report of a stillborn with a and nuchal cord x 2 and true knot, Dr. M. Maneschi (Universitat di Palermo, Sicili) in 1977 recommended that “a situation of this kind could be suspected on the basis of certain clinical findings to be elicited by questioning the patient about the intensity of active fetal movements, as

well as in the presence of changes of position of the fetus and the findings of a high presenting part at term. If on the basis of these findings the cord is suspected to be short, either in itself or as a result of coils around the fetal neck or body and if low placental insertion and other causes of the high position of the presented part have been excluded by echography (ultrasound) further tests can be carried out.”

Recently, Dr. N. V. Strizkova, Chair of Department of Obstetrics and Gynecology, Moscow Institute, and Dr. S. M. Petrikovsky, Obstetrical Professor, University of New York, used endoscopic devices in 1981 to visualize fetuses in the uterus with cord entanglement during labor. Their solution was helpful in determining fetal risk once the membranes were ruptured.

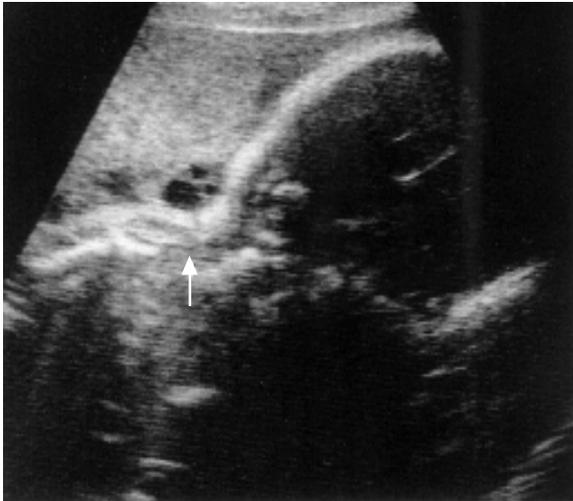
This attempt to manage the umbilical cord was followed by several scientific reports from China, Europe, Japan, and the United States, demonstrating ultrasound could visualize the umbilical cord. Specifically, in 1982 P. Jouppila, M.D. (Oulu, Finland) described the ability to visualize the umbilical cord around the fetal neck. “Antepartum diagnosis of cord coilings would be of clinical value for the adequate suppression and management of this complication,” he wrote.

As ultrasound technology improved, more and more anecdotal reports appeared. Today it is recognized that ultrasound can visualize cord entanglement, abnormalities, hematomas, varixes, knots, and architecture. In a 1989 review by Dr. Ingo Clausen, Professor of Obstetrics and Gynecology, University of Aarhus, Denmark, structural anomalies of the umbilical cord were mentioned as being diagnosed with ultrasound. Worldwide recognition that these potentially dangerous mechanical and acquired cord complications are responsible for antenatal fetal deaths are encouraging in that a solution and means of management are possible.

Ultrasound visualization of cord position, structure, and function can be displayed in several forms. Black and white (gray scale) ultrasound can identify the outline (edges) of the cord and reveal the arteries and vein. Comparison to the fetus and placenta can detail both attachments and position. Doppler color ultrasound can visualize blood flow in color and outline the cord in red, blue, and yellow colors depending on the direction of the blood flow. The two techniques provide a clear picture of cord condition relative to the fetus. (Figures 45-49)

The first step in managing the umbilical cord is visualizing it, and it can be visualized as soon as it forms. Measurements of the umbilical cord can begin as early as six to eight weeks. Knowing the length of the umbilical cord over time can help diagnose short or long cords, and thereby anticipate difficulty with labor.

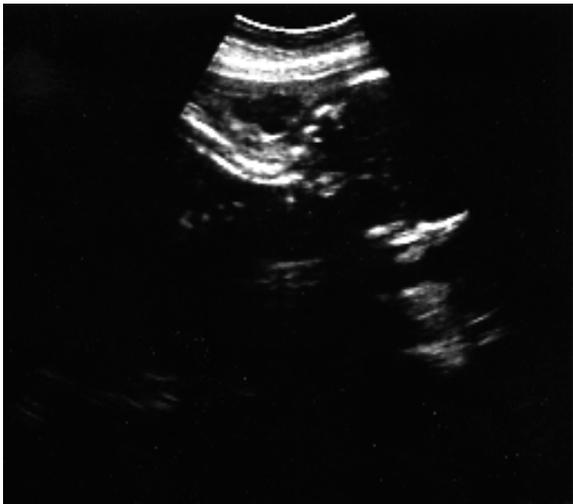
Umbilical and placental insertion sites and characteristics can be visualized with ultrasonography. In addition, it is possible to visualize an abnormal placental insertion or an umbilical cord



(Figure 45) Ultrasound: single nuchal cord.



(Figure 46) Ultrasound: double nuchal cord.



(Figure 47) Ultrasound: triple nuchal cord.



(Figure 48) Ultrasound: single nuchal cord Type A (cross over).



(Figure 49) Ultrasound: double nuchal cord.

defect. The diagnoses of these cords have been described in many writings to date. Knowing that an abnormal insertion site exists may direct the obstetrician to look for growth disturbances.

In 1995 Dr. Torgrim Sornes (Akerhusus Central Hospital, Norway) suggested that “there is an association between cord encirclement and fetal weight deviations, independent of cord length (effects)—an intense follow up should be done on these fetuses.”

Another observation of cord abnormalities and fetal effects by Dr. John Rolschau (Odense, Denmark, 1978) states, “It has been documented that a thin umbilical cord is associated with low birth weight.” Also, “The RNA/DNA ratio was increased, indicating tissue stress (in the placenta) in cases of battledore insertions of the cord.”

Few studies have observed large numbers of infants from conception to delivery with these findings. This is necessary if the effects are to be accurately determined. A means of determining prenatally entangled fetuses having difficulty is needed. Case reports have been published on an individual basis where fetuses were prenatally observed with ultrasound and delivered when believed compromised. Torsion can be recognized by measuring the distance between the veins (pitch). A close pitch suggests a multiple twists and the need to pay close attention to the fetus. Knowing which fetuses have the potential for umbilical cord complications is the next most important means of management is counting fetal movements.

The mother can play an important role in preventing a tragic loss from a cord accident. While it is unknown how much time is needed for a fetus to die, it is believed that in most instances the fetus dies slowly. As described earlier, fetal behavior is consistent and, if recognized by the mother, changes in fetal movement patterns can be documented and reported to the physician. Dr. E. Sadosky studied movement among fetuses with cord complications and felt there was a pattern to signs of fetal stress. Older literature describes decreasing activity of the fetus over time.

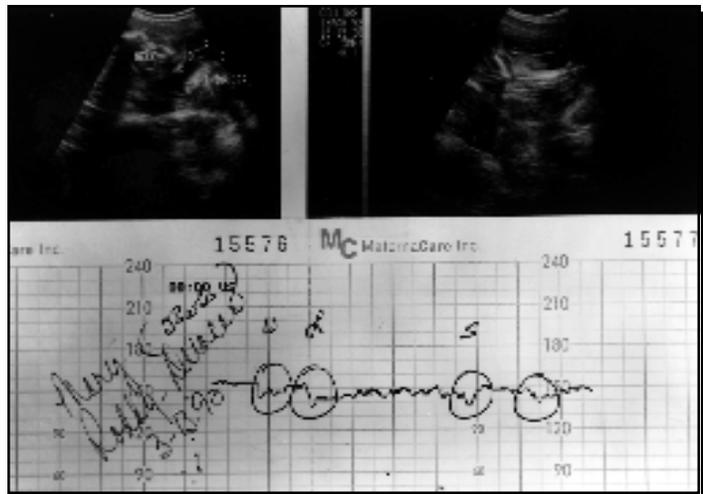
It is important for the mother to recognize when the fetus plays and when it sleeps. These activities should remain similar from 32 weeks until 40 weeks. In addition, fetal strength should remain similar.

However, fetal movement decreasing over time and weaker movement over time suggest fetal stress and should alert the mother to see her physician. Fetal jerking movement over time should also alert the mother. In addition, daily hiccup behavior (more than 4 episodes in 24 hours) suggest cord compression and initiation of fetal reflexes. These signs of probable fetal entanglement and/or placental insertion/location abnormalities should motivate the physician and mother to begin a surveillance plan, especially prior to 40 weeks. All detections of changes in fetal behavior noted by the mother or the physician should be shared with each other.

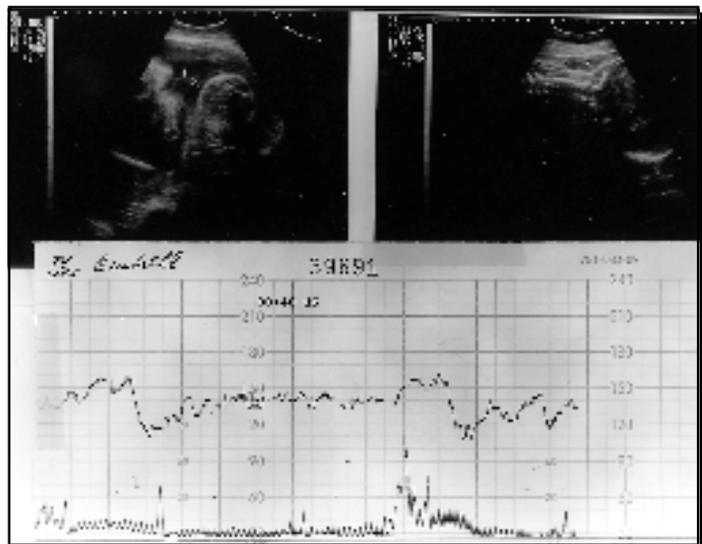
The second step is to measure (quantitate) fetal behavioral change. The simplest way is to evaluate the fetal heart rate, or FHR. FHR recordings of 15-minute strips can be invaluable during prenatal visits. Comparison over time can help identify a stressed fetus. The most sensitive indicator of fetal physiology is the heart rate pattern and characteristics. Fetal heart rate patterns change immediately with cord compression. Degree and duration of cord compression are unique. These patterns (called Lambda signs, V shape, W shape, U shape, variable decels, late decels, and overshoot) indicate fetal blood flow disruption. Patterns which repeat themselves and develop frequently suggest a compromised fetus. These pregnancies can be identified and studied. One method is to evaluate the FHR at home at least once a week with a fetal monitor. This may allow the discovery of a slowly deteriorating pattern which indicates delivery. Potential delivery is suggested with the detection of a double nuchal cord, an abnormal FHR pattern at 38 weeks, and a change in movement patterns for 24 hours. (Figures 50-52)

Meconium

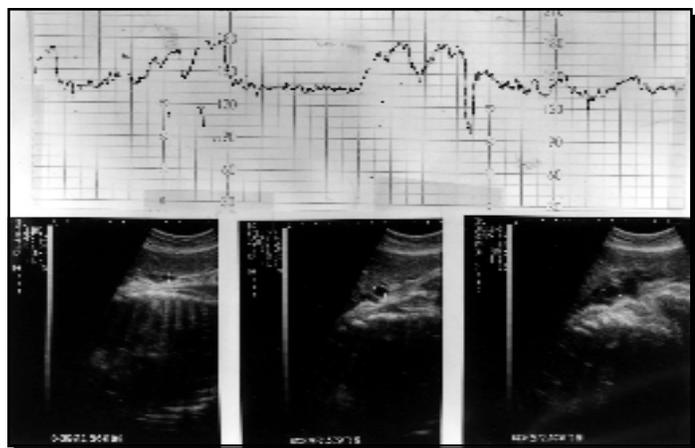
One of many concerns for



(Figure 50) Single nuchal cord showing L, W, V signs.



(Figure 51) Double nuchal cord with large Lambda decelerations on fetal heart rate recording.



(Figure 52) Umbilical cord with body loop with fetal heart rate decelerations.

such a fetus is the prevention of meconium effects. It is well known that by 36 to 38 weeks, the fetal colon is capable of peristalsis [movement] and contains bile acids for digestion, debris from swallowing vernix/lanugo hair, and cells. This intestinal mixture is called meconium. It is believed that the fetus expels this material which discolors amniotic fluid to the well known green color when "motilin," an intestinal hormone, is released. The stimulus for the fetus to expel, or have a bowel movement, in the uterus is thought to be due to fetal stress. It is believed that 1/5 of all deliveries contain meconium. Whether or not this is correct, it is believed that the fetus is at risk under these circumstances. Meconium, whether thick or thin (diluted or concentrated), is damaging to fetal lungs if inhaled. It is also damaging to fetal membranes, and can penetrate the membrane layers in one hour. If able to penetrate the amniotic barrier, cells called macrophages can transport these acids to the umbilical cord and can cause umbilical vessel constriction.

Experiments have demonstrated that umbilical arteries will be affected by meconium by stimulating the circular smooth muscle to tighten, thus closing the vessel off. This effect is observed to leave evidence of tissue damage as well as possible fetal circulation disturbances.

Because meconium has the potential to lead to fetal compromise, it seems reasonable to avoid it. It is also possible that cord compression can cause release of meconium by stimulating the release of motilin, creating additional cord vessel constriction and blood flow interruption. This argues for close monitoring of fetuses with cord entanglement.

The presence of meconium at birth does not mean or insure that fetal damage has occurred. According to Dr. Naeye's study of the Collaborative Project, only 0.2% of the meconium-stained amniotic fluids examined were attributable to birth asphyxial disorders. However, Dr. Naeye stated: "after all of the antecedent risk factors had been taken into account, meconium in the amniotic fluid had only one unfavorable pregnancy outcome, the presence of neurological abnormalities at 7 years of age. These neurologic abnormalities were of three types: quadriplegic cerebral palsy, lesser motor disorders with associated severe mental retardation, and chronic seizure disorders. These neurologic abnormalities are likely to be the consequence of vasoconstrictive effects of the meconium."

A management goal, therefore, is to anticipate the fetus at risk and deliver it before a meconium accident. The timing may be difficult since it is believed that some fetuses can release meconium in small amounts and that it can be reabsorbed within 24 hours. There is debate about whether this is so, but it is known that the older the fetus, the more likely the occurrence. Fetuses are currently allowed to reach what is termed post-maturity. Yet fetuses older than a gestational age of 42 weeks are considered post dates and are at risk of meconium.

A management approach would be to consider any fetus with cord entanglement by its due

date of 40 weeks needs to be delivered. Doing so may avoid meconium in these infants, as well as reducing stress factors to an already stressed state. In the Perinatal Umbilical Cord Project (PUCP), over 1,000 fetuses have been screened, and no fetuses with obvious cord problems delivered past 40 weeks. None of these fetuses had meconium at delivery, suggesting that meconium is avoidable. Ten percent of deliveries in the United States are associated with meconium. At least 1% have meconium complications. Fetuses prenatally diagnosed with umbilical cord complications should be aggressively managed to avoid meconium, and delivery should occur by 40 weeks.

Unless this issue is proven unimportant by future scientific studies, assuming that meconium is not dangerous seems risky. To date, no such study has been done.

Managing a fetus identified with an umbilical cord risk eventually requires a decision to deliver the baby. Reaching that decision can be difficult especially where the fetus is early. Prior to the delivery date, a series of evaluations are necessary to document the physiological state of the fetus. The avoidance of a premature delivery is the concern of the obstetrician or care giver.

Collecting information on how to decide when to deliver a compromised fetus sometimes depends on information derived within the hospital. The fetus can be studied in more detail and its movements assessed with ultrasound. For example, its amniotic fluid can be measured to look for decreased urine output. If the fetus is not making urine, it could suggest it is weak and therefore ready to deliver even though it is not in labor. The fetus' blood pressure can be indirectly checked with color Doppler flow measurements. Its average heart rate and reactions to induced contractions (oxytocin stress test) can be assessed. If it is abnormal, delivery can be initiated.

Tools are available to manage umbilical cord related fetal changes. Ultrasound, fetal monitoring, and fetal movement counts are all now available to evaluate fetal health. Currently, no prenatal blood test can be simply performed. Some tests can remove blood from the fetus by amniocentesis and indicate if delivery is necessary. These tests are risky and may themselves cause labor and delivery.

Two tests are interesting to discuss. One is called a "blood gas;" the other is called "erythropoietin." A blood gas is an absolute measurement of the fetal well-being. The fetus' oxygen level, pH level, and related chemistries such as Base excess can be quickly determined. A needle must be used to remove blood from the umbilical vein, requiring transabdominal/uterine penetration. If the blood shows acidosis (a low pH), it suggests the fetus is in need of delivery.

A more recently developed test which measures fetal reaction to oxygen levels is erythropoietin, a hormone specific to red blood cell production. Preliminary tests suggest that an elevated erythropoietin level means the fetus needs oxygen and should be delivered if the level is sus-

tained. Erythropoietin may also play a role in protecting neurologic tissue as it has been identified in the brain. Newborns with increased red blood cell production may show changes in the blood count test. Nucleated red blood cells appear as a result of the increased erythropoietin stimulus. These are immature cells and many appearing on a test suggest a closer look at the fetus.

None of these tests individually can decide whether a fetus should be prematurely removed from the uterus. Taken together, they form a solid base from which clinical judgment still must decide intervention based on experience. Management of newborns with cord accident related births goes beyond labor. Several tests are important to consider if a compromised fetus is delivered.

A third step to consider is a premature infant which is showing signs of stress from an umbilical cord problem. Situations such as monoamniotic twins have been managed where Betamethasone was administered by 31 weeks. This was done to mature the fetal lungs in the event of early delivery after 32 weeks. There are now several examples published where this was done. A case study of a single fetus with a double nuchal cord and true knot was managed this way and is presented at the end of this book.

Morbidity - Other Effects

Nuchal cord births have been observed to be predisposed to anemia. Not all nuchal cord births are the same, but “tight” nuchal cords seem predisposed to producing anemia in the newborn. In one study, 18.5% of nuchal cords were anemic. The National Collaborative Perinatal Project defined perinatal anemia as a capillary hematocrit of less than 40%. - Hgb < 13.2 g/1 Hct < 39.1%.

“The information derived from this study suggests the need for hemoglobin and hematocrit determinations on all neonates who have nuchal cord [complications]. If symptomatic at delivery, hematologic evaluation should be done promptly and the neonate examined for evidence of hypovolemia and hypotension. If asymptomatic, hemoglobin and hematocrit determinations should be done before discharge from the nursery in order to identify those neonates who are anemic.”

Angela J. Shepherd
Department of Pediatrics
University of Texas, Galveston
AJDC Vol 139, 1/1985 pg 71

Nuchal cord deliveries may also cause loss of fetal blood volume which, if greater than 20%, will induce “hypovolaemic shock”. This is a condition where the fetus has lost too much

blood. Observations have been reported where blood transfusions were necessary to correct this emergency. The clinical picture is different from lack of oxygen in that blood volume is missing but oxygen levels are normal. The newborn is usually characteristically pale, tachycardic with irregular respirations, and weak with low blood pressure.

This physiologic state is caused by decreased perfusion of tissue rather than an absence of oxygen and nutrients. The metabolic activity of the tissues remains static, so the chemical activity has nowhere to go - like a stagnant pond. Byproducts therefore have no place to go and oxygen, although available, cannot enter the cell. The result is a buildup of waste products and carbon dioxide which results in cellular "acidosis." This dangerous state can cause death if not corrected. Once discovered hypovolemia is quickly treated with fluids, plasma, and blood transfusion.

These events are not common but do happen. Indeed, knowing that the potential for morbidity exists can be enough to quickly recognize this clinical emergency. Newborns with cord problems should be reviewed for morbidity.

Blood also contains cells which react to tissue damage called white blood cells (WBC). Many types of white blood cells are called by various names, depending on the cell purpose. A polymorphonuclear leukocyte (WBC/PMN) is a cell very important in protecting tissues from bacteria and other invaders. It is also important in reacting to tissue which has been damaged by changes in blood flow. Tissues which line blood vessels such as umbilical arteries and vein are called endothelial cells and under high microscopic magnification look like ceramic tiles. These cells contain chemicals which, when released, can react with PMNs and incur damage. These reactions over time can affect the smaller vessels of the placenta or elsewhere and eventually contribute to organ dysfunction.

Not only does the fetus react as a whole to cord compression, and as a biologic agent on a cellular level, it also reacts on a molecular level. A rapidly emerging field in the medical sciences is one of molecular research. It is the step up from genetics and the step below cellular activity, but all are interrelated.

A possible marker for tissue damage is another WBC called a lymphocyte. Recent research by Dr. Naeye suggests that compromised fetuses that develop neurologic injury enough to cause cerebral palsy have changes in their lymphocyte count. When reviewing records of a group of CP infants, lymphocyte counts of greater than 10,000 were noted. These elevations did not occur in any less severe circumstances and did not recur. The changes seem important when determining when neurologic injury took place. The timing of stillbirth will be discussed based on the appearance of the infant. These concepts of forensic studies are important if scientists

are going to understand how umbilical cord accidents affect the fetus over time.

What lies in the future is speculative, but now a solution is possible. How will future umbilical cord complicated pregnancies be managed?

Non-stress Test:

“Our results suggest that the non-stress test should be used as the primary test for antepartum detection of fetal acidosis because it has the highest sensitivity; in addition, it is simple and easy to perform.”

Anthony M. Vintzileos, Chairman
University of Connecticut Health
Farmington, Connecticut
1991

¹ Antecedents of Cerebral Palsy, Karin B. Nelson, M.D. Jonas H. Ellenberg, Ph.D., National Institute of Neurological and Communicative Disorders and Stroke.

² Translation by Dr. Jeng Deng, Professor of Ultrasound, University of Wuhan, Wuhan, China, 1994.

Chapter 6: The Future of Umbilical Cord Research

“The goals of prenatal research must be to prevent cerebral palsy, to improve our understanding of prematurity, to give every baby its right to a sound body and mind when she or he is born, and perhaps one day make the NICU as obsolete as the iron lung [for polio].”

**Peter W. Nathanielsz, M.D., Ph.D., Sc.D.
Ithaca, New York 1995**

“Where there is no vision, the people perish.”

(Proverbs 29:18a KJV)

What can be expected in the future of research into umbilical cord complications? To conduct the research, resources must be obtained to pay for the sophisticated technologies and supplies needed. Trained personnel such as nurse midwives, medical statisticians, obstetricians, pathologists, and engineers will be needed, as well as competitive salaries. A review by the Institute of Medicine published in 1992 states that obstetrics and gynecology lacks supporters in government research agencies. In its survey it is estimated that “six to eight physicians/scientists each year are recipients of major training support. Only nine out of 250 departments of obstetrics and gynecology nationally received more than \$2 million in federal funds in 1990.” On one federal advisory committee, it was noted that there was a scarcity of obstetrics and gynecology representation even though a major source of hospital admissions to most acute care hospitals is from ob/gyn. Overall, the share of research funds from federal sources going to departments of obstetrics and gynecology has remained at a steady 1.5%.

Given adequate funding, the solution to umbilical cord complications is reachable. It appears possible that one solution to cord entanglement is to reach into the uterus and reverse it. Today, Dr. Michael Harrison at the University of California, San Francisco, enters the uterus as early as 24 weeks gestational age to repair holes in fetal diaphragms. This deadly defect, which occurs in about 1 of every 2,200 births, has been successfully treated this way.



(Figure 53) Stillborn male 36 weeks due to a nuchal cord twice around the neck.

Surgical manipulation of the uterus and fetus is highly complex and very risky. The demonstration that it can be done is equivalent to the Wright Brothers' first flight. The steps necessary to surgically repair diaphragmatic hernias are more involved than the steps it would take to untangle a fetus identified at risk, an event that may be many times more common than 1/2,200 for diaphragmatic hernia or 1/600 for mongolism risk. Every umbilical cord abnormality places the fetus at risk for stillbirth. This may be a 1/500 risk. Fetal harm without stillbirth may be 1/50 risk and unrecognized.

Entering the uterus with an endoscope smaller than the diameter of a pencil is possible. By comparison to a uterine incision the endoscope is significantly smaller. It essentially avoids trauma. Over the last decade this experiment has been performed in animals and humans with success. This suggests that a fetus imaged with 3-D ultrasound and diagnosed with entanglement at 24 weeks can be identified. Fetal heart rate changes compatible with cord compression can be visually evaluated and a fetus at risk for complications followed.

Today, needles are used to sample the umbilical cord for blood in infants with stress and illnesses. These invasive procedures are successful and proven to save fetuses' lives. A "hook wire" is inserted into the uterus to manipulate the cord. Several recent reports have used small gauge devices to manipulate the cord, puncture it, and suture it. One report described the use of this technique to obstruct the cord of a dead twin who was taking blood flow from a live twin.

All these efforts taken together say that entering the uterus with several small diameter devices to visualize the fetus, identify the cord, remove the entanglement and leave the fetus to heal and grow in the uterus is possible today. Reports from the Netherlands have demonstrated successful visualization of the fetus with an endoscope in monkeys which does not disturb the mother or create premature labor.

To accomplish the goal of fetal surgery for cord entanglement, funds are needed to design the equipment and to work with an established animal model laboratory with trained technicians and certification. To purchase a research monkey for study and its necessary support costs at least \$2,000 per monkey. This type of research is absolutely necessary to demonstrate to governmental authorities such as the Food and Drug Administration that these surgical techniques are safe and effective and can avoid stillbirth. Without this documentation, approval of these techniques will not take place.

Another approach being evaluated by the Pregnancy Institute uses Fetal Heart Rate monitoring. Currently being studied is a surveillance approach that depends on telemetry (remote telephone monitoring). Selected patients with cord entangled fetuses are equipped with special moni-



(Figure 54) Previous Figure 53 showing a pathologic double nuchal cord.

tors at home. These home fetal heart rate monitors are used by the mother to record fetal heart rate strips for telephone transmission. At pre-scheduled times the monitoring episodes are watched real time by the physician at home. This allows the patient to be home and allows the physician to choose a convenient time. Surveillance with telemetry based fetal heart rate monitoring is done every night until delivery, usually 36 to 40 weeks. It has been done as early as 28 weeks for 2 months straight. This clinical trial is still ongoing, and its objective is to demonstrate the feasibility of the approach. To date the study has been successful.

Stillbirth

“In non-malformed babies of birth weight greater than or equal to 2.51 kg— these babies are apparently well equipped to survive until the terminal event [normal but dead] and on the basis of this study there is likely to be a high degree of avoidability to their deaths.”

Bill Kirkup, M.D.
University of Newcastle
Medical School
1990

Statistical understanding of umbilical cord related stillbirths is inaccurate at best. Future efforts to more accurately depict events leading to stillbirth are needed. Publications from New Delhi, Norway, China, Scotland, Germany, Britain, and the United States cannot accurately determine how many antenatal or neonatal deaths are due to the umbilical cord. Because of inaccurate reporting by death certificates state by state, the National Center for Health Statistics cannot determine the extent of this mortality.

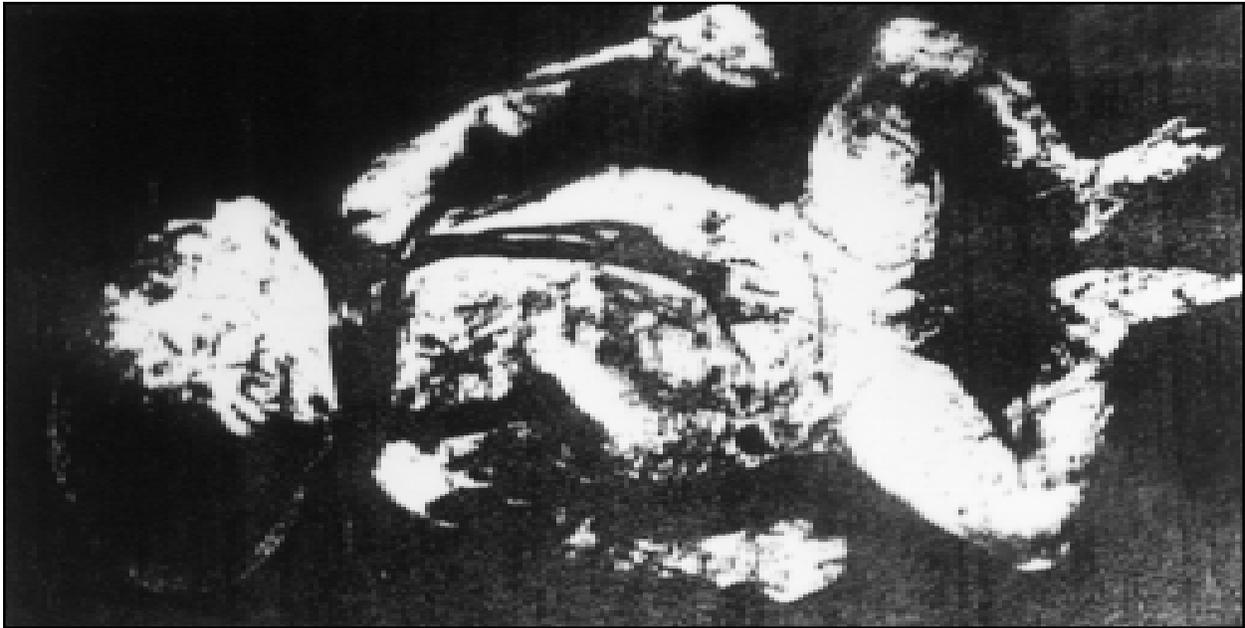
Fetal harm (morbidity) is completely unknown and unavailable. One Chinese study suggested morbidity consisted of neurologic damage and neonatal deaths. What worldwide statistics exist are largely based on labor and delivery events and not prenatal events. One study reviewed perinatal deaths in normal infants and related lack of antenatal care by 20 weeks as an important risk factor. In 30% of deaths there was an avoidable factor. In 61% of these cases, intrapartum management appeared to be an important factor. The most important aspect of management was recognition of fetal heart rate monitoring abnormalities. Therefore, many of the avoidable factors detected related to the recognition of probable fetal distress by fetal heart rate monitoring. In



*(Figure 55) Life Magazine, July 1969
Showing Dr. Edward Hon receiving fetal
heart rate patterns of cord compression.*



*(Figure 56) Life Magazine
Showing delivered infant
(breech delivery) with tight
single nuchal cord.*



(Figure 57) European fetus with triple nuchal cord – 38 weeks.



(Figure 58) Chinese fetus with triple nuchal cord - 38 weeks.

addition to these findings, some of these heart rate abnormalities arose 3 hours before death and did not resolve or remit but went unattended.

“Abnormal Fetal Heart Rate Patterns may be present for many minutes before actual fetal death.— Changes in fetal heart rates associated with umbilical cord compression may be progressive over many minutes and hence useful.”

**Edward H. Hon
Los Angeles, California
1975**

Estimating the time of death in stillborns is important in order to understand factors causing death. What events lead to the death of a fetus especially when the supply line is involved? In normal no-risk (no illness) pregnancies, this insight would be invaluable. A large review of cord-related stillbirths needs to be done to determine the timing of death. Is it random, circadian, nightly, daily during maternal activity, rest, after meals, etc.?

“Here again there had been recognition of life within 4 days of delivery – indicating that the fatal compression occurred with the advent of labor. – The mother had been uneasy about diminished foetal movements although the foetal heart was still distinctly heard; then within 2 days, the foetal heart disappeared.”

**T.F. Corkill
Wellington, New Zealand
1961**

Some insights about the timing of death may be discoloration of the umbilical stump, desquamation (loss of skin) of the face, abdomen or back; brown skin discoloration, and mummification. These changes correspond to 6, 12, 24 hours or to great changes from death to delivery. Reviewing stillbirths for these changes may help pinpoint time of death and fetal symptoms perceived by the mother.



(Figure 59) Vagina; delivery: Term infant of reduced weight with single nuchal cord.



(Figure 60) Vaginal Delivery: Term infant overweight with single nuchal cord.

Numerous examples have been published describing isolated case reports of maternal symptoms related to the cord mechanism involved. Reports include statements such as “pulling” sensations from the cord insertion site to “great” movements of the fetus as it changes position, or a “stick on a picket fence” as the abdomen of the mother is stimulated with fetal repositioning. Fetal jerking movements and hiccups are not uncommon complaints with nuchal cords and knots. “Rolling” movements are detected by the mother. Even “seizures” have been described. The most important symptom is “decreased fetal movement” sometimes days before death. We continue to tell patients that the fetus should not “slow down” toward term. The mother needs to be aware of fetal behavior. Patients can “feel” what the fetus is telling them. Every symptom and subtle sign should be used to suspect fetal compromise.

In European countries intrauterine fetal death (IUFD) is responsible for more than half of the perinatal mortality rate. The intrauterine fetal death rate reflects the complete failure of pregnancy surveillance and antenatal care. Therefore, the development of specific strategies in order to further reduce stillbirths becomes more and more important. More than 1/3 of stillbirths reported in Europe seemed predictable and avoidable by appropriate management after a positive fetal surveillance test.

More than 50% of the stillbirths occurred after the 34th week of gestation, when any infant has a nearly 100% chance to survive with modern pediatric care. At the same time, a marked decrease in the number of antenatal visits and quality of antenatal surveillance can be observed in cases of antepartum stillbirth. It should be concluded that antenatal surveillance has to be performed more intensively, especially in the last trimester.

“Intrauterine fetal death is the worst result of antenatal care for the obstetrician and creates tremendous grief with parents in the knowledge that they have lost an infant with full potentials for later life.”

Karl T. M. Scheider, M.D.

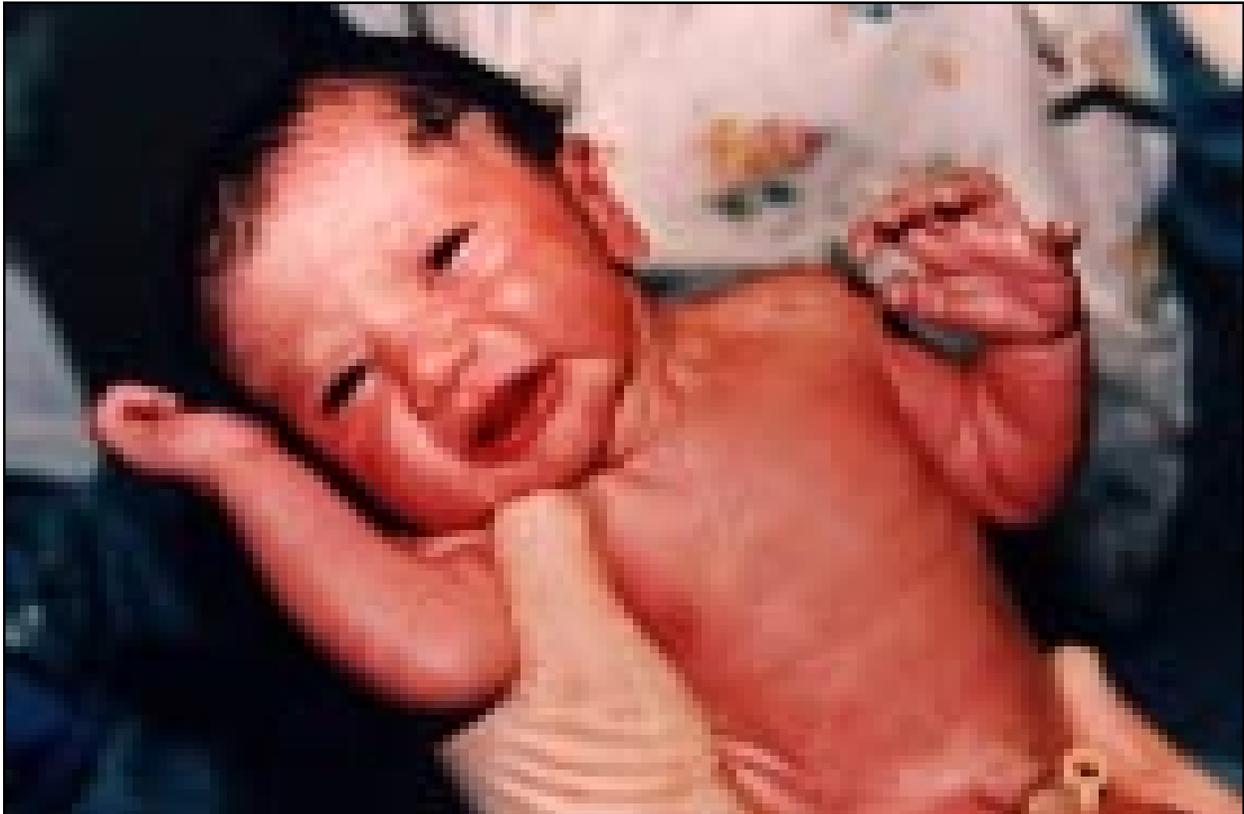
Munich, Germany

1994

Two types of deaths seem to be related to cord complications: sudden death, such as with cord rupture, hematoma, prolapse, and maybe entanglement; and slow death, such as with nuchal cords, knots, torsion, body loops. There are early deaths and late deaths which may have different patterns.



(Figure 61) 36 week fetus with decreased fetal movement and fetal heart rate changes. Delivered by C/Section; showing torsion/single nuchal cord/knee (r) loop. Note color and flaccidness of the newborn.



(Figure 62) Same fetus 15 minutes after delivery. Now 5 years old and without obvious complications.

Management of patients facing this potential harm will require different strategies. Non-invasive approaches will use ultrasonography to discern the fetus at risk. Future evolution of techniques using chemical factors from the maternal blood may be useful in following that specific fetus. Detecting fetal chemical changes from the maternal blood sample may allow the evaluation of a slowly dying fetus and the decision to intervene with delivery if late, or to intervene with manipulation of the fetus if early.

One older manipulation is external version. With computer assistance, knowing that the nuchal cord is entangled right to left, the fetus might be manipulated to unwind the loop. This is done by using one's hands on the pregnant abdomen and maneuvering the fetus around head to toe. These potential management techniques will have to be tried to determine their feasibility.

To imagine this maneuver, compare it to the game of 3-D movements inside a round frame. Like the game, the procedure could be used to slowly rotate the mother to induce counter movements in the fetus, thus untangling it. Although this may seem farfetched, with computer guidance and imaging it may be possible.

Another possible insight may be derived from NASA's Ames Research Center. NASA and specialists from the University of San Francisco's Fetal Treatment Center are developing devices which would be placed inside the uterus with the fetus. These devices would measure fetal vital signs. The intrauterine space becomes a tailored NICU. The biotransmitters send vital information by radio signal to be evaluated by computers outside the fetus. Already in existence is a thermometer capsule which is swallowed by astronauts to measure core body temperature. NASA co-developed this capsule which sends constant temperature readings to a monitor by radio signal. The ability to continuously know fetal blood pressure, heart rate, EKG patterns, and oxygen/pH status will be important. Ultimately, it will be necessary to know glucose/lactic acid/catecholamine erythropoitin and cortisol/ACTH levels. These chemistries provide insights into the fetal condition and would assist in deciding the need for delivery. For instance, a cord entangled fetus with decreased movement, low oxygen levels and elevated erythropoitin might need delivery.

Current fetal blood tests attempting to determine time of injury only determine damage already done. These tests include measuring fetal lymphocyte counts over time. In the future, it may be possible to detect early changes as the damage is beginning and rescue the fetus before irreparable harm occurs.

Umbilical cord complications can be solved just as polio was solved. It is important to do so because the fetuses are normal. They are not defective, malformed, or irreparably injured. What is required is support to create the solutions. The solutions are at hand.

“At present, we can offer no single solution to the problem of umbilical cord complications which remains probably the most important cause of fetal depression at birth.”

Samuel Bruce, M.D.

New York, NY

1978

“By means of a model similar to that of Clapp, et al we have reported evidence of an increase in free oxygen radicals as possible causal factor in cerebral lesion. These observations (of umbilical cord compression/blood flow reduction) should alert physicians to pursue more investigative studies on the potentially ominous FHR pattern in relation to the long term outcome of the infants, particularly that of neurologic performance.”

Yuji Murata

Edward J. Quilligan, 1994

(Editor-in-Chief of American Journal of Obstetrics and Gynecology)

CASE STUDIES

I) A Difficult Case for Prevention A Combination Umbilical Cord Accident

This term pregnancy in a 28 year old G3 P1 AB1, was uneventful until one weekend before the due date. Fetal movement ceased after a day long fishing trip. Evaluation on labor and delivery indicated a fetal demise. Spontaneous vaginal delivery revealed a stillborn male fetus (7 1/2 lbs.) with three nuchal cords, a shoulder loop-body loop, a two vessel cord, straight architecture and a true knot. In addition to these findings was a cord length of 100 cm and marginal placental insertion. Which abnormality caused death is disputed in the medical literature. When and how these fetuses die is difficult to document. Whether diet plays a role or maternal activity changes uterine physiology is unclear. The Perinatal Umbilical Cord Project (PUCP) seeks to address the problem of umbilical cord accidents. (Figure 63)



(Figure 63)

II) Time of Death: Nuchal Cord Type B and Clinical Clues

A 30 year old white female G1 P0 was doing well prenatally up until 28 weeks when the fetus was noted to be breech turned from vertex. A single nuchal cord was diagnosed and fetal heart rate changes were normal range. Re-exam at 30 weeks confirmed a breech lie with a (NCX1) and normal FHR. A scheduled return in two weeks and expected repositioning of the fetus to vertex was pre-empted by stillbirth 2 days before the revisit. Saturday night the patient noted fetal movement of normal strength and duration at 10:00 p.m. The fetus was breech and did not exhibit “jerking” movements or hiccups suggesting no stress during the breech lie.

Awakening at 7:00 a.m. on Monday morning, no movement was felt as expected. An office visit confirmed fetal demise and an ultrasound showed a triple nuchal cord and a vertex lie. At delivery, a normal male (6 lb.) had a triple NCX3 with a type B NCX1 (see text). Also seen was a marginal cord insertion into the placenta. (Figures 64-68). Microscopic exam of the placenta showed chorangiosis (a placental change associated with asphyxia). The Perinatal Umbilical Cord Project (PUCP) has observed there are two types of nuchal cords (A & B) and that these stillbirths may occur at night during sleep.



(Figure 64)



(Figure 65)



(Figure 66)



(Figure 67)



(Figure 68)

III) Fetal Heart Rate Changes: Warning Signs of Cord Compression

Over 1,000 pregnancy cases have been reviewed for cord complications in the Perinatal Umbilical Cord Project. What has repeated itself over and over are the fetal heart rate patterns associated with cord compression. Depending on the degree of compression, duration, intermittency, and gestational age of the fetus – fetal heart rate responses tell us a lot about this event prior to labor. Three cases briefly described here suggest a variety of presentations.

Case KD a 30 year old G3 P2 with normal deliveries previously. This fetus developed a shoulder loop and fetal heart rate changes prior to labor (Figures 69 & 70) shows the fetal heart rate during a NST

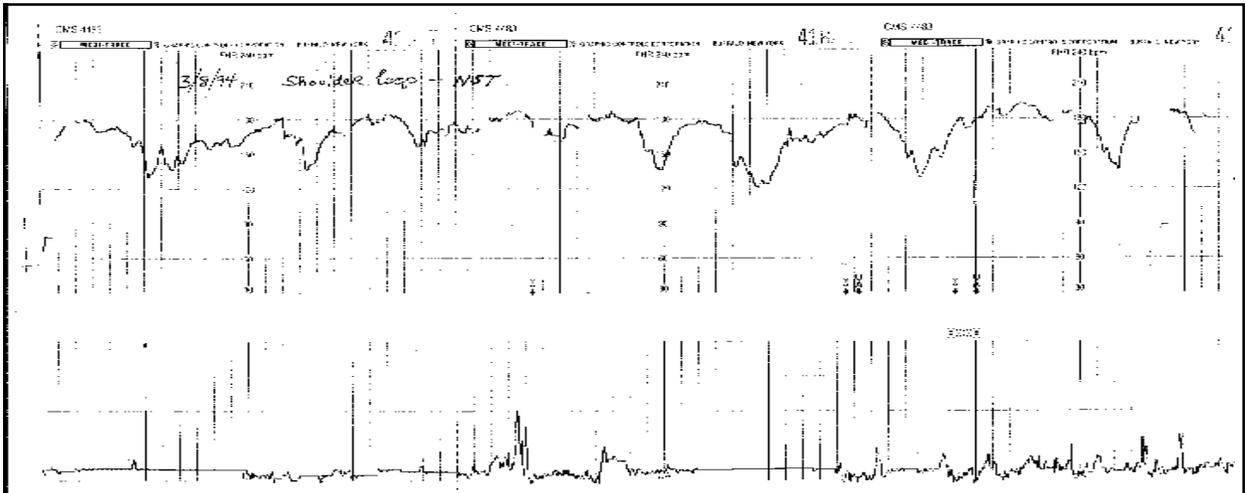
FHR strips 3/8 - 3/9 (Figures 71 & 72) and subsequent resolution of the shoulder loop during a next day OCT. Delivery was uneventful, but the newborn had a persistent ductus arteriosis and the placental exam showed chorangioma (Figure 73). At five (5) years old the young girl is developing normally.



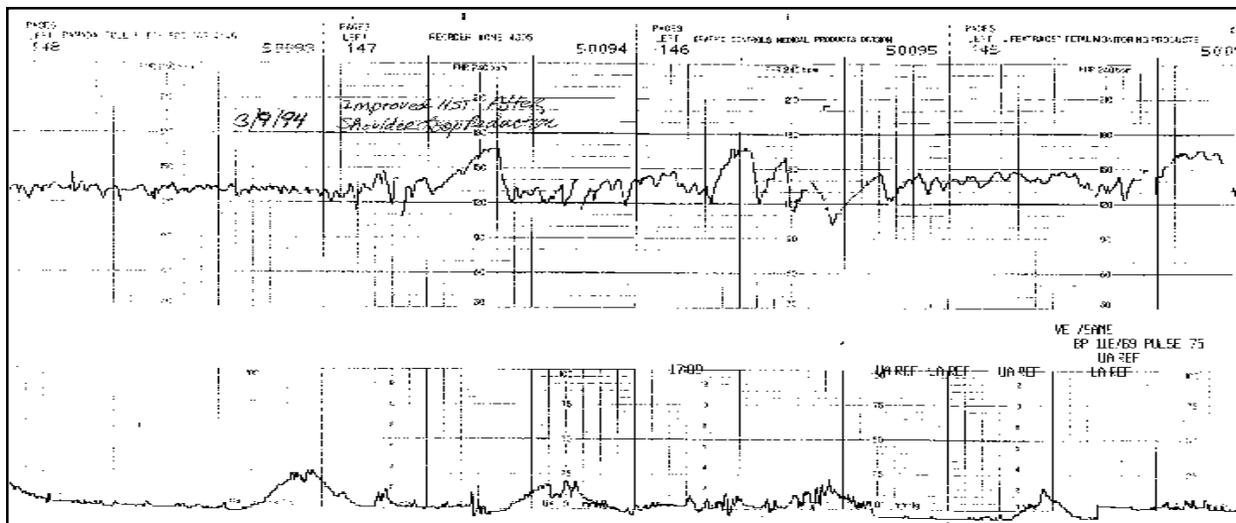
(Figure 69)



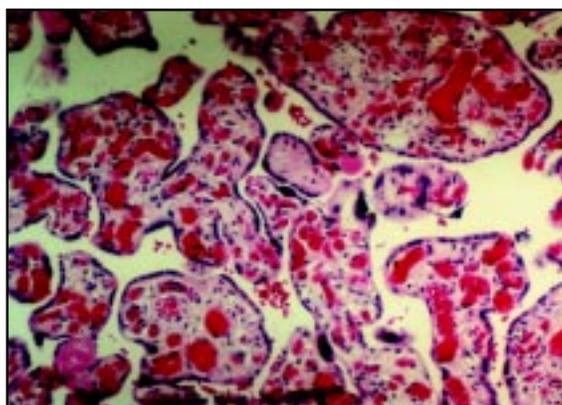
(Figure 70)



(Figure 71) FHR strip 3/8.

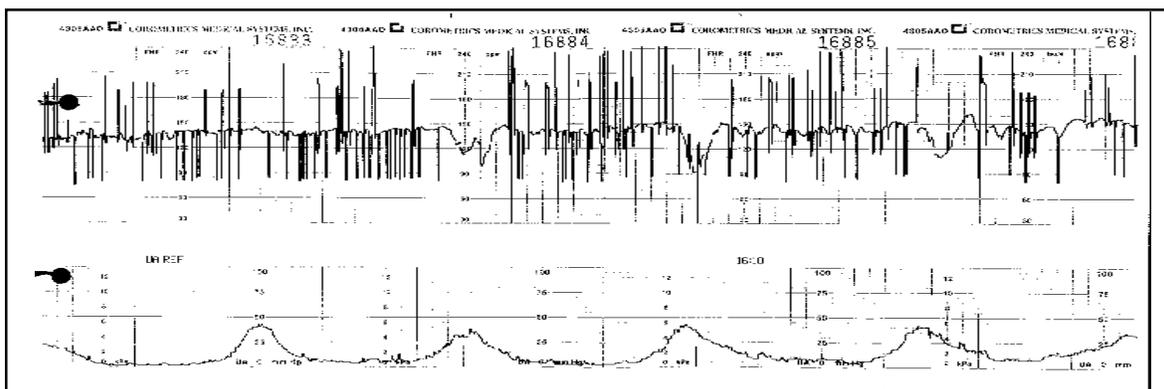


(Figure 72) FHR strip 3/9.



(Figure 73) Chorangiosis

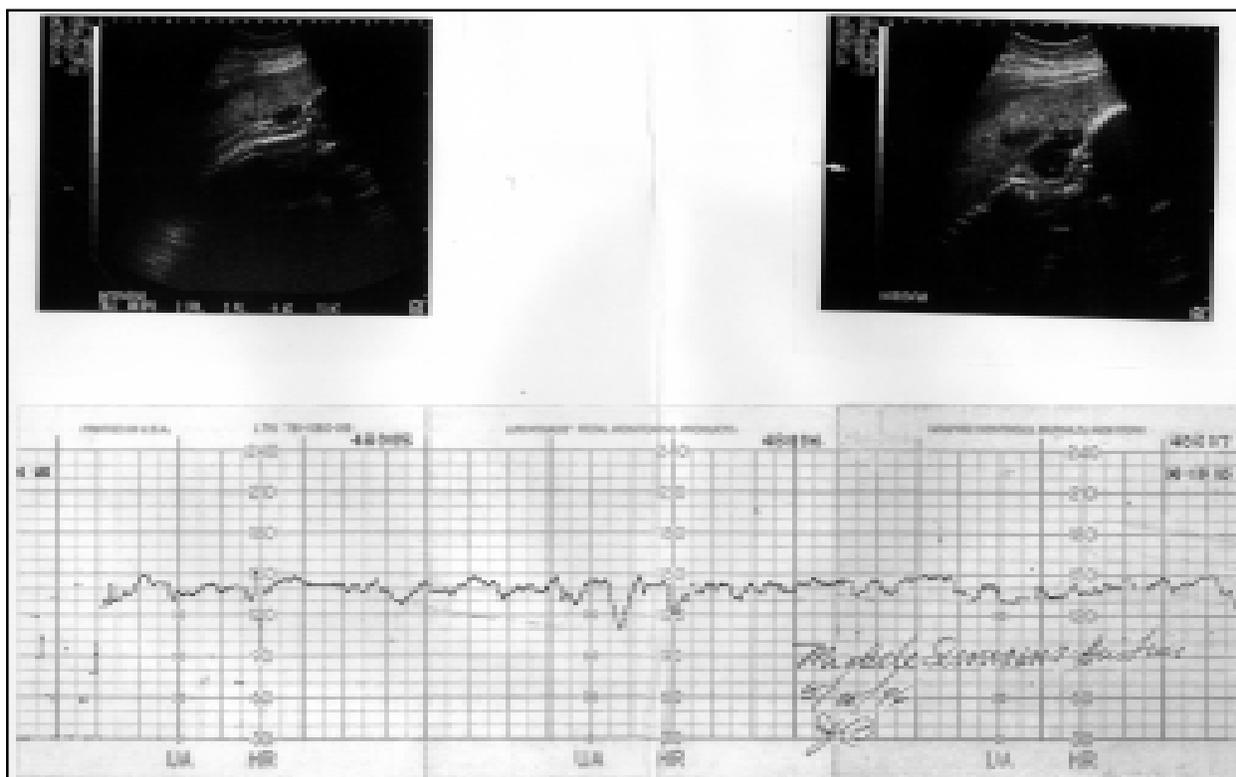
Case TR a 35 year old G4 P3 developed an arrhythmia during labor. An intermittent arrhythmia was noticed prenatally. As labor progressed, the arrhythmia became more pronounced. Eventually the FHR became non-reassuring causing a C/Section. A posterior placenta with marginal/sacral umbilical insertion was present. The newborn EKG was abnormal for 2 hours. Cause unknown. (Figure 74).



(Figure 74)

IV) Umbilical Cord Risk Managed After A Previous Cord Accident

Case MS 27 year old G2 P0 SB1, had a previous stillbirth associated with a double nuchal cord. Her subsequent pregnancy was monitored with frequent ultrasounds and FHR monitoring. At 28 weeks a double nuchal cord was seen on ultrasound. At 32 weeks a true knot was verified. Delivery by 36 weeks confirmed a double nuchal cord with true knot. The patient correctly recognized decreased fetal movement and changes in the fetal heart rate (on home monitoring) (Figures 77- 79).



(Figure 77) Top: 28 weeks ultr-sound showing double nuchal cord and cord bunching. Bottom: Fetal heart rate recording at 28 weeks with spike variable and W sign suggesting cord compression.



(Figure 78) Double nuchal cord.



(Figure 79) True knot.

V) Umbilical Cord Risk Managed After A Previous Cord Accident With Subsequent Morbidity

TF 28 year old G3 P1 SB1, was being monitored for repeat cord risk. Her previous stillbirth was due to a double nuchal cord and triple knot at 36 weeks (Figures 77-79).

During the 3rd pregnancy a nuchal cord was diagnosed by 32 weeks and followed to 36 weeks where decreased fetal movement and fetal heart rate variables prompted delivery. At 48 hours the infant developed persistent pulmonary hypertension and required NICU care and oscillator support for one week. At six months the newborn is well.



(Figure 77) Stillborn

Cord compression may be associated with pulmonary hypoxia due to shunts delivering blood flow to the adrenal gland, heart and brain. Animal model studies have suggested this connection. Recent computer cord blood flow models also suggest loss of pulmonary blood flow during fetal stress, hypotension and low cord flow. Pulmonary injury may not be uncommon.



(Figure 78) Double nuchal cord



(Figure 79) Triple knot

VI) Umbilical Cord Torsion, A Not Uncommon Occurrence in Newborns

Umbilical cord torsion is usually not diagnosed in a live born. 10% of deliveries may have uncomplicated torsion. Torsion can coexist with constriction, a different pathology altogether. Amniotic bands may also be present with both the former pathologies. (Figures 80-82) are examples of torsion, constriction and umbilical cord bands.



(Figure 80) Straight cord with twists (torsion).



(Figure 81) Umbilical cord torsion with constriction at the umbilicus.



(Figure 82) Umbilical cord torsion, constriction and amniotic band in a 24 week fetus.

SUMMARY

Umbilical cord accidents can be avoided. The solution requires intensive observation and identification of the fetus at risk for this silent complication. Once identified, fetal heart rate monitoring, fetal behavior monitoring and maternal awareness of the problem will be the best chance of achieving a live birth. With an estimated 4,000 deaths per year from umbilical cord complications, an initial goal of 50% reduction is possible. Finally, defining the morbidity of cord compression is the next major goal.

-ARTICLES OF NOTE-

"Watching the Unborn Inside the Womb: High Risk Mothers and the Graph that Raises Their Babies' Chances", Edward Hon, M.D., Life Magazine, July 25, 1969 pages 63-66.

"The Umbilical Cord", Samuel R. Reynolds, Scientific American, July 1952, Volume 187, page 70.

"Le Cordon Ombilical (Fonis Umbilicalis)", L. Arvy et G. Pillepi, Editors, Ostermundigen, 1976, Switzerland.

"Today's Latest Coo - Deborah Norville's Delivery", (which includes an umbilical cord knot), People (weekly), March 25, 1991, pages 67-73.

"Death Before Life" - Kevin Kanty, An Account of Stillbirth due to Umbilical Cord Torsion," Vogue Magazine, May 1995, pages 93-96.

"Ann Jillian's Miracle Baby - Delivery Room Drama" (with a triple nuchal cord discovered at C-section), National Enquirer, February 25, 1992.

"The Infant's Vulnerable Life-Line", T.F. Corkill, M.D., Australian New Zealand Journal of Obstetrics and Gynecology, 1961, Volume 1, Page 154.

"Ecologic Physiology of the Fetus, Ultrasonography of Supply-Line Deprivations Syndromes", Jason C. Birnholz, M.D., Radiologic Clinics of North America, 1990, Volume 28, Number 1 page 179.

"Intrapartum Ultrasound Diagnosis of Nuchal Cord as a Decisive Factor In Management", Steven J. Feinstein, M.D., et al, American Journal of Obstetrics and Gynecology, 1985, Volume 153, page 308.

"Non-Coiled Umbilical Cord Vessels: A New Marker for the Fetus at Risk", Tom H. Strong, M.D., Obstetrics and Gynecology, 1993, Volume 81, page 409.

"Decreased Fetal Movements Associated with Umbilical Cord Complications", E. Sadovsky, M.D., et al, Israel Journal of Medical Science, 1977, Volume 13, page 295.

"Pregnancy Events and Brain Damage", Leon Mann, M.D., American Journal of Obstetrics and Gynecology, 1986, Volume 155, page 6.

"Brain Damage After Intermittent Partial Cord Occlusion in the Chronically Instrumented Fetal Lamb", James F. Clapp III, M.D., et al, American Journal of Obstetrics and Gynecology, 1988, Volume 159, page 504.

"Variable Fetal Heart Rate Decelerations and Electrocardiac Activities", Yuji Murata, et al, American Journal of Obstetrics and Gynecology, 1994, Volume 170, page 689.

"Brief Repeated Umbilical Cord Occlusions Cause Sustained Cytotoxic Cerebral Edema and Focal Infarcts in Near-Term Fetal Lambs", Harmen H. De Hann, et al, Pediatric Research, 1997, Volume 41, page 96.

ADDITIONAL RECOMMENDED READING

A Child is Born, Lennart Nilsson, Text by Lars Hamberger, Bantam Doubleday Dell Publishing Group, Inc., 1993, ISBN 0-440-50691-3.

Behavior of the Fetus, William P. Smotherman and Scott R. Robinson, Telford Press, Caldwell, NJ, 1988.

Disorders of the Placenta, Fetus, and Neonate: Diagnosis and Clinical Significance, Richard L. Naeye, M.D., Mosby Year Book, St. Louis, MO, 1992.

Fetal Behavior: Developmental and Perinatal Aspects, Jan G. Nijhuis, Oxford University Press, Oxford, England, 1992.

Fetal Cells in Maternal Blood: Prospects for Noninvasive Prenatal Diagnosis, Joe Leigh Simpson and Sherman Elias Eds., New York Academy of Sciences, New York, New York, 1994.

Fetal Monitoring Interpretation, Miki L. Cabaniss M.D., editor, J.B. Lippincott Co., Philadelphia, PA, 1993.

Forging a Women's Health Research Agenda: Policy Issues for the 1990's, Jeri A. Sechzer, Anne Griffin, and Sheila Pfaffin Eds., Annals of the New York Academy of Sciences, Vol. 736, 1994.

Invasive Fetal Testing and Treatment, Christopher R. Harman, M.D., Editor, Blackwell Scientific Publications, Winnipeg, Canada, 1995, ISBN 0-86542-208-7.

Life Before Birth and a Time to be Born, Peter W. Nathanielsz, M.D., Promethean Press, Ithica, NY, 1992.

The Newborn Infant: One Brain for Life, Claudine Amiel-Tison and Ann Stewart Editors, Published by the French National Institute of Health and Medical Research, Paris, France, 1994.

Pathology of the Human Placenta, Kurt Benirschke and Peter Kaufman, Springer-verlag, New York, New York, 2nd edition, 1990.

Reproduction in Man and His Ancestors for 700 Million Years, Richard Torpin, M.D., McGowen Printing Company, 1974.

Strengthening Research in Academic Ob/Gyn Departments, Jessica Townsend Editor, National Academy Press, Washington, D.C., 1992.

When a Baby Dies: The Experience of Late Miscarriage, Stillbirth, and Neonatal Death, Nancy Kohner and Alix Henley, Harper Collins Publishers, San Francisco, CA, 1995, ISBN 004-440-934-6.

