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New Zealand College of Midwives [Inc]

NEWSLETTER

Volume 5, Number 1 : January 1992

Remits SGM

Sex After Childbirth

MIDIRS

NEW ZEALAND COLLEGE OF MIDWIVES INC.

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Place of Work _____

Type of Membership

Full member (Registered Midwife Full or Part Time)	$74.00
Full member (Student Midwife or Registered Midwife on Maternity Leave or Unwaged)	$37.00
Associate Member (Other Interested Individual)	$74.00
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Please tick your choice of payment method.

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Date of Birth _____ NZNA member: Yes/No

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Place of Work _____

Please return completed form (together with money, if applicable) to your local Treasurer.

RESEARCH: THE EDUCATION OF MIDWIVES IN NEW ZEALAND

I hope to have completed this project early in December, and as I near its completion, I would like to take this opportunity to thank all those who responded to the questionnaires and for the wide range of thought and suggestion that your responses encompassed.

I have completed an analysis of the data and am now in the process of writing an account of the midwifery services and education that are described within it. It has been a pleasure to have been able to do this research and to share your experience, insights and the general optimism about the work and learning that you have embarked upon together.

The overall impression is one of women and practitioners clear about relationships and expectations which are grounded in mutual respect and confidence in the abilities of each other, and about the goals which they work towards.

Thankyou all for your help
Yours sincerely

Natali Allen

Natali Allen

NEWS & VIEWS

Trust you have all had a pleasant, relaxing holiday period and are looking forward to the anticipated busy year ahead.

Direct Entry Midwifery

Congratulations Auckland Technical Institute and Otago Polytechnic Direct Entry Midwifery Courses. These two courses start in 1992.

Commiserations to Carrington and Wellington. It is very exciting to at last have Direct Entry Midwifery Education underway. Both programmes will require continuing support from our midwifery community.

Midwifery Education Workshop

This workshop will be held on 12th, 13th and 14th February at Victoria University Nursing Studies Room, Wellington. The workshop is to formulate guidelines for areas such as Protocols/Referrals, Post Graduate Degrees, Code of Practice, Accreditation, Obstetric Regulations and Service Delivery.

Any ideas or information you have on any aspects of the above topics please contact your local Chairperson.

Health Minister Meeting

Representatives of NZCOMI will be attending a meeting with the Health Minister, Simon Upton, on the 4th February to discuss issues relating to Primary Health Care and Independent Midwifery Practice.

Maternity Benefits

Discussions with the Department of Health re Maternity Benefits are ongoing, with the next meeting set for the 4th of February.

Carey Virtue and Ron Lovell have compiled the data from the Questionnaires sent out in August-September. A big thank you to them both. Thank you all who have contributed.

Nurses and Midwives Bill

A draft of the Nurses and Midwives Bill has been received by NZCOMI. The first consultation takes place in Wellington on 6th March with the Minister of Health.

- See you all at the Special General Meeting, 14th February.
- Conference dates have changed – see following advertisement.

BOM

FUTURE EVENTS

26-27 March 1992:	*ICM Regional Meeting* – Melbourne, Australia.
14-17 June 1992:	*10th International Congress of Psychosomatic Obstetrics and Gynaecology* – "Reproductive Life" Contact: Congress Secretariat, CONGREX, International Society of Psychosomatic Obstetrics and Gynaecology (ISPOG) - 92, PO Box 5619, S-114 86 Stockholm, Sweden. Tel: 46 8 32 69 00. Fax: 46 8 32 62 92.
4-7 October 1992:	*Second International Homebirth Conference* Sydney, Australia. Reclaiming our Heritage, Creating our Future. Contact: Conference Secretariat, GPO Box 2609, Sydney, NSW 2001, Australia. Tel: (02) 241 1478, (02) 247 6940. Fax: (02) 251 3552.
1-4 November 1992:	*The Fourth International Conference for Maternity Nurse Researchers* – Taipei, Taiwan, The Republic of China Contact: Yueh-chih Chen, RPN, PhD, Director, School of Nursing, College of Medicine, National Taiwan University, 1, Jen-Ai Road, Section 1, Taipei (10018), Taiwan, The Republic of China.
9-14 May 1993:	*International Confederation of Midwives 23rd International Congress* Vancouver, BC, Canada.

MEDIA WATCH

NZ Herald
27/12/91

MIDWIVES ENJOY GAIN IN STATUS

NZPA Wellington

Approval for two experimental direct entry training programmes shows that midwifery is being accepted as a profession in its own right, says the president of the College of Midwives, Karen Guilliland.

Midwives had battled for years to establish their position as independent health professionals, she said.

The Nurses Amendment Act 1990 allowed them to practise independently of hospitals or doctors.

The Associate Minister of Health, Katherine O'Regan, gave approval this week for the Otago Polytechnic and the Auckland Technical Institute to offer three-year, direct entry midwifery courses in 1992 and 1993.

Each course would take up to 16 students, and provide training in general medicine and surgery, but with emphasis on women's health.

Until now, midwifery training involved a three-year general nursing course followed by specialist training. Prospective midwives spent a lot of time studying subjects that had no bearing upon childbirth.

Opioids in labour are on the come-back trail

A combination of opioid and local anaesthetic administered by the epidural route improves the quality of analgesia in labour and because small doses of each are used decreases the side effects associated with epidural analgesia. "Opioids in labour are making a comeback".

About 40 per cent of mothers choose epidural anaesthesia for pain relief in labour because it is highly effective. However, it is not without its drawbacks. Though almost all experience at least considerable relief of pain some complain of persisting perineal sensation. Higher concentrations of local anaesthetic sometimes produce a motor block in the legs which can be uncomfortable, and hypotension is an occasional problem.

A useful alternative is finding favour. This consists of a mixture of local anaesthetic and opioid by the epidural route. Alone, opioids do not provide very good analgesia during labour and systemic absorption results in sedation of both mother and newborn baby, nausea, pruritus and respiratory depression. Pethidine is probably the pick of the opioids since it has a weak local anaesthetic action but analgesia from a single dose is unpredictable and local anaesthetic top ups are often required.

Opioids and local anaesthetics are synergistic and the combination seems to work favourably in obstetrics. Fentanyl, a pethidine derivative, is short acting and must be given as an infusion or else can be added to bupivicaine top ups. In this way the dose of bupivicaine can be halved without loss of pain relief while motor block in the legs is less of a problem. Other benefits include better relief of perineal pain, more rapid onset and prolongation of analgesia and less likelihood of hypotension and perhaps instrumental delivery. Other opioids or derivatives that have been tried successfully include diamorphine and a new agent sufentanil. Both improve analgesia when given with bupivicaine.

Side effects still occur with the combination: pruritus and nausea are quite common but are less of a problem with lower doses. Opioids cross the placenta but when given by the epidural route do not seem to adversely affect the Apgar score, though more sensitive tests may show neurobehavioural changes. Systemic and epidural opioids must never be given concomitantly because of the risk of respiratory depression.

Although there are a "few reservations the judicious use of opioids and local anaesthetics in combination can improve analgesia for labouring mothers even further".

Lancet 337:1446-1447, 15 Jun 1991

SPECIAL GENERAL MEETING

Friday, 14 February 1992, at 6.30pm

Wellington Polytechnic

School of Nursing and Health Studies

Restructuring of NZCOM to be voted on.

Remits included in this Newsletter.

All members are encouraged to attend.

For further information or sharing of ideas please contact your local Chairperson.

New Zealand College of Midwives Conference

28, 29, 30 August 1992 Wellington

Research has shown that CONTINUITY of midwifery care results in a lower intervention rate and greater consumer satisfaction with birth experiences.

The 1990 Nurses Amendment Act acknowledges a woman's right to CHOOSE her care-givers.

The CHALLENGE is developing a service that reflects:

* Partnership with women
* Knowledge that allows women real choice
* Strength and unity within our profession

CALL FOR ABSTRACTS

We welcome prospective participants to submit abstracts of up to 250 words, for papers addressing the concepts of continuity, choice, and challenge in midwifery practice. Typed abstracts and speaker profile required. **Closing date 28 February 1992.**

Abstracts and inquiries to: **Janet Lambie, 18 Fortification Road, Wellington 6003, New Zealand.**

Telephone: (04) 388-7090

Facsimile: (04) 388-2887 IDD: (64 4) 3882-887

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arteries of the placenta. Prolonged vasoconstriction can lead to the obliteration of some of these vessels (Giles et al, 1985; McCowan et al, 1987).

It is thus clear that most cases of 'placental insufficiency' are, in reality, examples of maternal vascular insufficiency resulting from inadequate placentation. It may appear pedantic to draw this distinction but the continuing use of the term 'placental insufficiency' leads to a false emphasis being placed upon functionally unimporant abnormalities of the placenta, diverts research along unrewarding pathways, and obscures the fact that many complications of the later stages of pregnancy have their origin in the early months of gestation.

Acknowledgements

Figure 3 is reproduced from *Pathology of the Placenta* by H Fox and is reproduced by kind permission of W B Saunders and Company.

Figures 1, 2 and 4 are reproduced from *Basic Science in Obstetrics and Gynaecology* by J Dewhurst, M De Swiet and G V P Chamberlain and are reproduced by permission of the editors and Churchill Livingstone.

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EMFs and Breast Cancer

Matthew Connelly and Louis Slesin

In 1989, Genevieve Matanoski of Johns Hopkins University reported one of the most intriguing findings in the EMF literature: a cluster of six cases of male breast cancer among 50,000 New York telephone workers.[1] Male breast cancer is extremely rare, with only 900 cases reported each year in the United States; so when Matanoski announced her extraordinary results, other researchers hastened to follow up.

Within six months, a team from the Fred Hutchinson Cancer Research Center in Seattle, Washington, had found six times the expected number of breast cancer cases among electricians, power station operators, and telephone linemen.[2] Then, in December 1990, Norwegian researchers reported a doubling in the overall incidence of male breast cancer among workers exposed to EMFs, with four times the expected rate among railroad and tram drivers.[3]

By 1991, interest in the subject had grown considerably. At a conference sponsored early in the year by the National Institute for Occupational Safety and Health, epidemiologists were unanimous in their support for a study of EMFs and female breast cancer. Two weeks before the conference, the American Cancer Society had warned that 1 in 9 women could expect to develop breast cancer—an increase over the 1987 rate of 1 in 10. And in a cover story in *Time*, a leading researcher declared that "something in our environment is contributing."[4]

While no one knows what is responsible for the soaring rate of breast cancer among women, several experts are beginning to turn their attention to EMFs. Funding, however, is a problem. Richard Stevens of Battelle Pacific Northwest Lab in Richland, Washington, has been trying for the past five years to raise money to test his hypothesis that EMFs are linked to the steadily increasing incidence of breast cancer—so far, without success.

Many years have passed since Nancy Wertheimer first discovered an association between EMF exposure and breast cancer.[5] In light of the strongly supportive evidence that has followed, we can no longer ignore her findings. Further study is nothing short of a high priority.

Notes

1. Genevieve Matanoski, Elizabeth Elliot, and Patrick Brysse, "Cancer Incidence in New York Telephone Workers," *The Lancet 337* (23 March 1991): 737.
2. Paul Demers et al., presented at the 23d Annual Meeting of the Society for Epidemiologic Research, Snowbird Resort, Utah (12–15 June 1990).
3. Tore Tynes and Aage Andersen, "Electromagnetic Fields and Male Breast Cancer," *The Lancet 336* (22 Dec 1990): 1596.
4. Claudia Wallis, "A Puzzling Plague: What Is It about the American Way of Life That Causes Breast Cancer?" *Time* (14 Jan 1991): 48–54.
5. Nancy Wertheimer and Ed Leeper, "Magnetic Field Exposure Related to Cancer Subtypes," *Annals of the New York Academy of Sciences 502* (2 July 1987): 43–54.

ARTICLES OF INTEREST

Sex after Children

If we define sexual relations as any expression of intimacy, then many things are possible.

William F. Van Wert

A woman called me recently. A former lover and long-time friend, she is now happily married, with a devoted husband and a new son. During the conversation, she confided that she had no sex drive left since the birth of her child, that she felt strangely asexual. She even joked that she was probably dysfunctional and something must be wrong. Her confession shocked me, because I remembered her as a passionate person, a fervent lover with an enormous sexual appetite.

Michael Weisbrot

A Common Refrain

My shock dissolved as I recalled the many similar comments I had heard from men—not about themselves, but about their wives. The lament goes something like this: "There's been nothing between us since the birth of the children" or "We're like strangers passing in the night" or "She won't do the things she used to do before" or "She's tired all the time" or "She won't let me get near her." It is as though the passion evaporates, the courtship rituals cease, the business of living overshadows the adventure of living together, a deep divide sets in, and an estrangement builds up. The many intimacies that two people share before birth give way to one new ingredient—the child—who not only "rules the roost," but becomes the main focus of the partnership.

They do the child, do for the child, talk about the child, listen for the child, lose sleep over the child, don't go out because of the child. All previous necessities—such as shopping, doing the laundry, bathing, eating, and resting—come to revolve around the child, and new priorities begin to crystallize. The parents spell each other. Some take turns on middle-of-the-night awakenings/changings/rockings; others take turns keeping daytime vigils on the high chair, the carseat, or the child-in-arms. Sex becomes an expendable option, a luxury to do without, a bother, a memory. Jobs matter; affording the baby matters. In this all-too-familiar scenario, physical intimacy between man and woman places a very distant third, at the most.

The woman who called was not calling to tell me secrets about life with her husband, with whom she is very much in love, or to flirt with former passions. Her confession came with a question: when we were lovers and I was a recent single father, how had I managed a sexual relationship while parenting three young children? She was unwilling to write it off as strictly a gender difference, and so was I—even though we both knew that soaring levels of prolactin, the "mothering hormone," were part of the picture.

For some reason, I remembered Sigmund Freud's account of instinctual renunciation. In *Civilization and Its Discontents*, he explains that the

© Mothering, no 60, Summer 1991, pp 115-117.

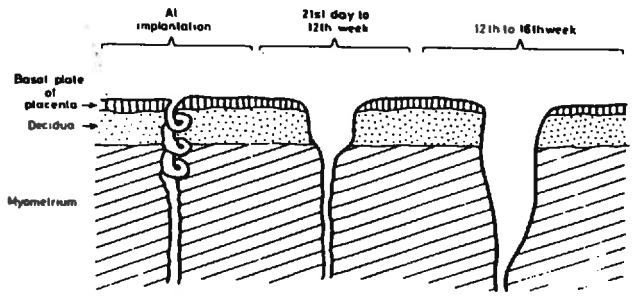

Fig. 4 Diagrammatic representation of conversion of spiral arteries in the placental bed into uteroplacental vessels. This takes place in two stages which results firstly in dilatation of the intradecidual portion of these vessels and, secondly, in the dilatation of the intramyometrial portion of these vessels

placental vessels (Fig. 5). This abnormality leads to a marked restriction of maternal uteroplacental blood flow and hence of the ability of the mother to supply oxygen and nutrients to the fetus. This accounts for all the fetal complications encountered in cases of pregnancy-induced hypertension, complications which are clearly a result of inadequate placentation.

It is now clear that this abnormality of placentation is not restricted to women who develop pregnancy-induced hypertension. A similar defective invasion of the placental bed spiral arteries by extravillous trophoblast is also a feature of most cases of idiopathic intrauterine fetal growth retardation in normotensive women (Robertson, 1981).

The haemodynamic consequences of inadequate placentation are now well defined. It has been known for a long time that maternal blood flow to the placenta is reduced in pregnancy-induced hypertension (Browne & Veall, 1953; Dixon et al, 1963). It is also clear that there is a similarly reduced blood flow in most cases of 'idiopathic' fetal growth retardation in normal women (Lunnell et al, 1979). A severe reduction of maternal blood flow to the placenta may not only restrict fetal growth but also result in under-oxygenation of the fetus. In these circumstances there may be changes in the fetal circulatory system in order to maximise blood flow to vital organs such as the brain. Fetal blood flow through the placenta is reduced as one component of the circulatory adaptation and this decrease, which can now be readily assessed by Doppler ultrasound (McParland & Pearce, 1988), is due to vasoconstriction of the fetal stem

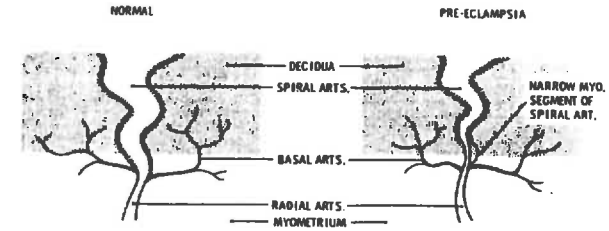

Fig. 5 Diagrammatic representation of the partial failure of placentation in patients destined to develop pre-eclampsia. In patients destined to develop pre-eclampsia the intramyometrial segment of the spiral arteries does not undergo physiological change and does not dilate, with consequent restriction of maternal ability to deliver nutrients and oxygen to the fetus.

PLACENTAL INSUFFICIENCY AND ABNORMAL PLACENTATION

It is still widely believed that a high proportion of cases of otherwise unexplained fetal death, hypoxia or growth retardation are due to a failure of the placenta to fulfil its physiological role of transferring oxygen and nutrients from the mother to the fetus. It has to be stressed, however, that this concept of 'placental insufficiency' is a purely clinical one for which a pathological basis has not, and cannot, be defined. It is becoming increasingly difficult to accept that placental failure, due to intrinsic placental damage, actually exists and it is increasingly clear that the common thread running through cases of presumed 'placental insufficiency' is a reduced maternal blood flow to the feto-placental unit. Only in recent years has the pathological basis for this circulatory inadequacy been clearly defined and in order to understand this vascular abnormality it is necessary to consider the process of placentation during the early stages of pregnancy.

During the early stages of gestation trophoblastic cells spread out from the placenta to colonise the decidua and adjacent myometrium of the placental bed, these cells being known as the 'extravillous trophoblast'. In addition these cells also stream into the lumens of the intradecidual portion of the spiral arteries of the placental bed. The intravascular extravillous trophoblastic cells destroy and replace the endothelium of these maternal vessels and then invade the media with resulting destruction of the medial muscular and elastic tissue (Brosens et al, 1967). The arterial wall becomes replaced by fibrinoid material which appears to be derived partly from fibrin in the maternal blood and partly from proteins secreted by the invading trophoblastic cells (de Wolf et al, 1973). This process is complete by the end of the first trimester, by which time these 'physiological' changes within the spiral arteries of the placental bed extend to the myometrio-decidual junction.

After this there is a 'rest phase' in this process but between the 14th and 16th weeks of gestation there is a resurgence of intravascular trophoblastic migration with a second wave of cells moving down into the intramyometrial segments of the sprial arteries, the cells extending as far as the origin of the spiral arteries from the radial vessels. Within the intramyometrial portion of the spiral arteries of the placental bed the same process as occurs in their intradecidual segment is repeated i.e. replacement of the endothelium, invasion and destruction of the medial musculoelastic tissue and fibrinoid change in the vessel wall.

The end result of this trophoblastic invasion of, and attack upon, the vessels of the placental bed is that the thick walled, muscular spiral arteries are convereted into flaccid sac-like uteroplacental vessels (Fig. 4). These can passively dilate in order to accommodate the greatly augmented blood flow through this vascular system which is required as pregnancy advances.

The extravillous trophoblastic cells therefore play a key role in placentation and through the activity of these cells the placenta establishes the adequacy of its own blood supply and ensures an ample supply of oxygen and nutrients to the fetus. The crucial importance of this process is shown by the finding that in women destined to develop pregnancy-induced hypertension in the later stages of their pregnancy there is a partial failure of placentation. This results in a markedly restricted maternal blood flow to the uteroplacental unit. This failure has two components. Firstly, in contrast to a normal pregnancy in which all the spiral arteries of the placental bed are invaded by extravillous trophoblast, this process occurs in only a proportion of these vessels in women who later develop pregnancy-induced hypertension, with a significant fraction of the vessels of the placental bed showing a complete absence of physiological change (Khong et al, 1986). Secondly, although the first phase of the arterial invasion by trophoblast occurs quite normally, with these cells evoking a physiological change in the intradecidual portion of the spiral arteries, there is a complete failure of the second stage; endovascular trophoblast failing to advance into the intramyometrial portion of these vessels (Robertson et al, 1967, 1975). As a result there is incomplete transformation of the spiral arteries into utero-

desire of the individual becomes sacrificed to conform to the will of the group. Whatever we call that group —the tribe, the community, or civilization itself—it permits the illusion of romantic love in coupling; it even fosters the provisional reality of couples standing apart and feeling apart. Why? To stave off anarchy and rebelliousness, and also to encourage the evolution of the couple to a family unit—the sum of which forms a group. The apparent dichotomy between couple and society serves to resolve the dichotomy at the level of the family, and thereby cement the social relations of the group. The point is that the survival of the family unit is dependent upon this deceit, seduction, and entrapment.

In other words, *to have a baby is to come out of privacy as a couple and to go public*—and, in the process, to be socially transformed by laws, institutions, and religions. The bad news is that the couple never regains its privacy. The good news is that it was only an illusion anyway.

At some surface or deep-down level, we feel "tricked" into the social order of parenting: tricked by others, tricked by each other, tricked by ourselves, even tricked by the newborn. But this deceit and the loss of privacy cannot alone account for the prolonged resumption of sexual relations or the radical altering of these relations.

For that, we must look to the couple. Much of what transpires between partners after birth is a direct consequence of how they interact during pregnancy. If they maintain physical relations (in the form of dancing, hand-holding, "making spoons" in sleep, and lots more), if they maintain communication (verbal intercourse in lieu of the physical variety, especially while in bed), if they maintain a sense of humor, mutual respect, honest consideration for each other, then the resumption of sexual relations need not be too traumatic. I know, easier said than done. Few of us have been blessed with such good fortune or foresight.

The Postpartum Rift

Couples with even the best intentions and behaviors during pregnancy still experience some rift after birth. If indeed, borrowing from my mother's recipe, sex is three parts mental (anticipatory) and one part physical (doing it), then the first three ingredients are already under siege, numbed by chronic fatigue, scheduling stresses, loss of sleep, and an overwhelming list of domestic tasks and labors (groceries, laundry, doctor visits, announcement cards, thank-you notes, and more). Then, too, the actual managing of the baby (feeding, changing, walking, burping, bathing, rocking, massaging) more than meets the touch/be-touched needs of most mothers and fathers.

The rift also becomes evident in the realm of emotions. The woman

Angel Molina

feels as though she has recently emerged from a physical possession, has a battered body she barely recognizes, is lumpy or misshapen, has been stretched and perhaps torn and sewn together again at the genitals, may leak from her breasts at any moment, and may choose to reserve her breasts for the baby alone. Feeling little or no self-esteem as a desirable sexual being, she sublimates her own desires into her new caretaker role.

The man, who may have already experienced some displacement during pregnancy, feels his malaise full force after birth, resenting the child for the endless disruptions, envying the child's on-demand access to his partner's body, and having urges that are now perceived as unreasonable. He may be temporarily put off by the woman's body ("Is *this* the body I impregnated?"), or he may be in awe of her Madonnalike qualities and sense a kind of mother-whore split, or he may sublimate his desires by throwing himself into work, drink, sports, or other forms of avoidance.

To hear couples tell the tale, the halt in sexual relations is usually the woman's fault. Men say they are ready, they have always been ready to resume, but the women have changed, feel too tired or ugly, won't tolerate the barest of touches, or reject them outright. Women blame themselves as well, having succumbed to culturally instilled guilt for failing to fit the role models shown in books, movies, and mass advertising. Unfortunately, society offers no sensitive portrayals of this crucial time.

As weeks pass, things sometimes go from bad to worse. Men feel tricked, as though everything in the coupling came down to this: the women used their men to get their children, and the men lost their women. Many men compound the problem by assuming martyrdom. Simultaneously, they fantasize, dream of escape, think of affairs, and begin to see their mother-in-law in the new mother. *What they fail to see is that the new mother has not only displaced her man for the child, but displaced herself as well. And what both partners fail to realize is that this displacement is natural, necessary, and temporary.*

The point is that both women and men participate in the rejections. The undoing of the couple, however, is not a fait accompli. Physiology alone will soon turn things around, especially among breastfeeding women. As the increased production of prolactin drops off, and as the menstrual cycle returns, so do sexual desires. But until then, there is work to do. And, with the stereotypical burden for the decline in sexual relations falling on the woman, on account of cultural codes and simulacra, the burden for redressing the situation falls on the man.

Healing the Rift

If we define sexual relations as genital sex alone, then redress may be next to impossible. But if we define sexual relations as any expression of intimacy, then many things are possible. The idea is not to preclude genital sex or endlessly defer it, but rather to place it in a larger context. What follows is a list-

ing of some possible intimacies.

Touching. Having children teaches us that much of our sexual urge is a longing to touch and be touched. Now is the time to rediscover handholding, hugging, and kissing—not as "foreplay" or, in old manual terminology, "necking and petting," but as intimacy. Heightening one's sensitivity to the many expressions of physical tenderness breaks old patterns and takes the charge out of genital sex, allowing it to return spontaneously. *Note:* Comparing the frequency of genital sex before parenthood with the frequency in the postpartum period is a poor gauge of intimacy.

Massaging. No special techniques are required, other than attentiveness to individual preferences and endurance levels. The more tired partner gets the massage; the less tired one gives it. Remain sensitive—if the so-called erogenous zones are too sore to be stroked comfortably, a hand and foot massage will feel wonderful. Massages of all sorts not only provide touch contact, but also instill trust and a new bonding between partners. Remember, massage is presented as a gifting, not a getting.

Sucking. When the new mother is engorged and leaking, and baby is asleep, the new father can suckle gently and get his own taste of breastmilk. But ask first; not all mothers like this form of intimacy.

Talking. Talk life, talk sex, talk sexuality. Share what sex has meant up to this point and how it feels now. This form of sharing creates an opportunity for new intimacy.

Providing accoutrements. Prepare good food for each other, listen to music together in the dark or by candlelight, fall asleep together in a refuge other than bed. Add special new touches of tenderness and mystery.

Forming a ménage à trois. With baby, that is. Falling asleep or taking a bath as a threesome is a powerful way of bonding and rediscovering the relationship. Most men operate on visual cues, and nakedness can create an erotic fulfillment in itself. Dressing and undressing together—with a bit of style—can be very satisfying as well.

Sharing with other couples. Informal discussions with friends, neighbors, and family about how they survived can take the edge off unmet needs. Conversing with others also invites input and insights that couples immersed in the situation may not be able to glean for themselves.

Stroking the ego. New mothers and fathers need their *egos* stroked as much as, if not more than, their bodily parts. A man wants to know he's still virile. His partner can let him know by "talking dirty" in his ear, or by watching him masturbate or helping him masturbate. Likewise, a woman wants to know she's still seductive, even though she may not think she is or may not choose to act on it. Talking, looking, and stroking apply here as well.

Embracing humor. Couples who develop a shared sense of humor—about how the baby runs their lives, or how little time or energy or sex they have together—keep open, and even expand upon, their means of communication. Naturally, humor needs to be genuinely shared, and not used as a barb or painful reminder of unmet needs.

The point is to be creative, while maintaining direct and honest contact. People stop having sex, fall out of love, and become estranged for a variety of reasons; but having children need not be one of them. The drought-after-children jokes we hear are culturally sanctioned ways in which men complain to one another about their women. Such jokes, although they overlook the male contribution to the problem, may be three parts funny and one part pathetic when the children are very young. But when the children are 10, 12, and 16, and the couple still has not resumed sexual intimacy, then the situation is no parts funny, very tragic, and professional therapy is probably necessary.

There is no crime in feeling nonsexual after a birth experience. The crime is in holding those nonsexual feelings to oneself, making lame excuses or telling lies to one's partner, and eventually killing the relationship. Whatever you do, don't throw out the parents with the bathwater.

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TEN STEPS TO SUCCESSFUL BREASTFEEDING

A Joint WHO/UNICEF Statement (1989)

Every facility providing maternity services and care for new-born infants should:

1. Have a written breastfeeding policy that is routinely communicated to all health-care staff.
2. Train all health-care staff in the skills necessary to implement this policy.
3. Inform all pregnant women about the benefits and management of breastfeeding.
4. Help mothers initiate breastfeeding within a half-hour of birth.
5. Show mothers how to breastfeed and how to maintain lactation even if they are separated from their infants.
6. Give new-born infants no food or drink other than breast milk unless medically indicated.
7. Practise rooming-in. Allow mothers and infants to stay together 24 hours a day.
8. Encourage breastfeeding on demand.
9. Give no artificial teats or pacifiers (also called dummies and soothers) to breastfeeding infants.
10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from hospital or clinic.

nature with only a small proportion of the villous population showing evidence of either an active or a healed inflammatory process. This degree of villitis is unlikely to impair, let alone dissipate, the functional reserve of the placenta. It seems unlikely, therefore, that the association between villitis and intrauterine growth retardation can be explained simply in terms of placental damage and the most probable basis for this relationship is that a villitis is usually an indication of fetal infection. There is a widespread belief that the placenta acts as a barrier to fetal infection but, in reality, virtually all organisms can breach the placental defences and infect the fetus. The placenta acts largely as a physical barrier which delays, rather than prevents, the passage of organisms to the fetus. The effects of fetal infection vary with the stage of gestation but infection during the later stages of pregnancy inhibits fetal DNA synthesis and thus impairs fetal growth.

The only obvious exception to this general rule is malaria. In endemic areas, malarial infection of the placenta is very common and is frequently associated with fetal growth retardation, transplacental passage of the parasite is nevertheless rare. In malaria there is, however, a massive accumulation of monocytic cells in the intervillous space and this is of a degree as to almost certainly impair maternal blood flow through the placenta (Galbraith et al, 1980).

Ascent of organisms from the birth canal, whether bacterial, viral or fungal, can lead to infection of the placental membranes — a chorioamnionitis. Infection of the membranes results in a loss of their normal glistening translucency and in severe cases the membranes may be opaque and foul smelling. Choriaomnionitis is commonly found in cases of prolonged membrane rupture, i.e. over 24 h, and is also frequently present in placentae from babies delivered prematurely, particularly those weighing less than 2.5 kg (Chellman & Rushton, 1985). There is a growing, but still not totally substantiated, belief that chorioamnionitis is an important aetiological factor in pre-term delivery (Drife, 1989), being capable of causing premature onset of labour, because of prostaglandins released from the inflamed membranes (Lopaz-Bernal et al, 1989), and premature rupture of the membranes.

TOXIC DAMAGE TO THE PLACENTA

Depressingly little is known about the possible injurious effects on the placenta of toxins, drugs or environmental pollutants. There is, for example, virtually nothing known about the effects on the placenta of excessive intake of alcohol or the use of addictive drugs. Indeed, the only example of toxic damage which has been adequately studied is that of the effects of maternal cigarette smoking. Interest in this topic has been stimulated by the well known association between smoking and low birth weight.

Placentae from women who smoke show changes suggestive of a reduced maternal blood flow. This is probably due to the vasoconstrictive effects of nicotine on the uterine musculature, for it is known that smoking a single cigarette causes an acute reduction in blood flow through the intervillous space (Lehtovirta & Forss, 1978). Electron microscopy of placentae from women smoking during pregnancy shows focal degenerative changes within the trophoblast which do not appear to be ischaemic in origin (van der Veen & Fox, 1982) and it has been suggested that this damage is due to cadmium. This substance is present in cigarette smoke and is specifically toxic to placental tissue (Di Sant' Agnese et al, 1983). Recently, Burton et al (1989) have described subtle changes in the villous fetal vessels of placentae from cigarette smokers which they thought could also be due to cadmium.

There is thus no doubt that the placenta is damaged by cigarette smoking. The degree of damage is, however, relatively slight and unlikely to dissipate the functional reserve of the organ. It is thus probable that the fetal growth deficit produced by cigarette smoking is due partly to nicotine-induced vasoconstriction of the uterine vessels, with a consequent inadequate maternal supply of nutrients, and partly to the direct effects of nicotine and carbon monoxide on the fetus.

can be considered as a manifestation of ageing (Fox, 1979).

Often adduced as evidence of placental ageing is the claim that placental growth and DNA synthesis cease at the 36th week of gestation (Winick et al, 1967). More recent studies have, however, shown that total placental DNA levels continue to rise in a linear fashion up to, and beyond, the 40th week of pregnancy (Sands & Dobbing, 1985). This finding is in accord with histological evidence of fresh villous growth in the term placenta (Fox, 1978) and with autoradiographic and cytophotometric studies which have shown continuing DNA synthesis in the villi of the placenta at term (Geier et al, 1975: Hustin et al, 1984: Iversen & Farsund, 1985). The continuing growth of the placenta has also been confirmed by morphometric techniques showing a continuing expansion of the villous surface area and progressive branching of the villous tree up to and past term (Boyd, 1984).

GROSS LESIONS OF THE PLACENTA

Few, if any, of the various plaques, thrombi and cysts seen on cutting the placenta are of any importance (Fox, 1978, 1986). Macroscopically visible calcification, however marked, is also a banal finding, devoid of any clinical significance.

It has already been remarked that lesions functionally inactivating a high proportion of the villous population are not accompanied by any ill-effects on the fetus and hence it appears paradoxical that infarction of more than 10 per cent of the villous parenchyma is, by general concensus, associated with a high incidence of fetal hypoxia, growth retardation and death (Fox, 1978). A fresh infarct is moderately firm and dark red; as it ages it becomes progressively harder and its colour changes progressively to brown, yellow and white. An old infarct thus appears as an amorphous, hard, white plaque. An infarct is due to thrombosis of a maternal utero-placental vessel and infarction of more than 10 per cent of the organ implies that there must be multiple thrombi in the maternal vasculature. This is a process which would not be expected to occur in a healthy vascular tree and it is therefore not surprising that infarction of a significant extent is virtually confined to placentae from women with pregnancy-induced hypertension. An acute atherosis is found in the uteroplacental vessels of women with pregnancy-induced hypertension (Fox, 1988), and the women are predisposed to develop thrombosis. Far more importantly, however, in women with pregnancy-induced hypertension, whether thrombosis occurs or not, there is a severely restricted maternal blood flow to the placenta (the reason for this is discussed later). Extensive infarction only occurs, therefore, in the setting of a severely compromised uteroplacental circulation and it is this which is the true cause of the apparent effects of infarction on fetal oxygenation and nutrition. Hence the true significance of extensive infarction is that it is the visible hallmark of an abnormal maternal vasculature and a severely compromised maternal blood flow to the placenta. The infarction is not the primary cause of the fetal complications and would be of no importance if it occurred in a placenta with a normal blood supply.

PLACENTAL INFECTION

Infections reaching the placental tissue from the maternal blood or from the endometrium, result in an inflammation of the villi — a villitis. A predominantly villous inflammation can be due to placental involvement in maternal infections such as rubella, toxoplasmosis, cytomegalovirus disease, syphilis or listeriosis. However such conditions account for only a very small proportion of cases of villitis, the vast majority of which are of unknown, or undetected, aetiology (Russell, 1987). Histological evidence of villitis is found in between 8% and 10% of all placentae in Western countries (Russell, 1980; Knox & Fox, 1984) and there is a clear correlation between villitis and intrauterine fetal growth retardation. Not uncommonly it has been assumed that the deficit in fetal growth is due to damage inflicted on the placenta by the inflammatory process. Most cases of villitis are, however, of a focal

Can we abandon Naegele's rule?

NIGEL SAUNDERS CATHERINE PATERSON

A pregnant woman may become anxious if she has not delivered by the date given to her by her physician. Much anxiety would be alleviated if a range of dates (38–42 weeks) was substituted for a specific date of delivery.[1]

Lancet 1991; 337: 600–01.

Introduction

"I cannot imagine why gestation should be the only process connected with reproduction for which a total exemption from any variation in its period should be claimed".[1]

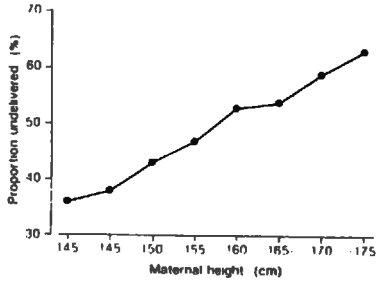
The duration of human pregnancy is a matter of considerable social and scientific importance. Both ancient Hindu and Roman cultures recognised that the average duration of pregnancy was about 9 months but allowed some latitude, especially with respect to prolonged gestation. In more modern times the legal process has been called upon to settle questions of legitimacy and inheritance in relation to the maximum possible duration of human pregnancy. For example, during the Gardner peerage trial (House of Lords, 1816), five eminent physicians testified that under no circumstances could the duration of pregnancy exceed 40 weeks' gestation, whereas five of their colleagues presented the opposite view.

The laws of some countries have defined an upper limit of gestation consistent with legitimate birth after the death or departure of a husband, whereas English law does not set a precise limit. In judicial practice, it is usual to accept the most recently agreed ruling and to inquire whether to go beyond it is reasonable.[2] The aim of the law is peace rather than justice; therefore, the scientific validity of such judgments is questionable.

Naegele's rule

Although most so-called prolonged pregnancies are due to delayed ovulation,[3] in clinical practice the time of ovulation is rarely known with certainty. Assessment of gestational age is helped by antenatal ultrasonography, but for most women an estimated date of delivery (EDD) is calculated according to Naegele's rule—ie, add 7 days and 9 months to the date of the last menstrual period. This formula, generally credited to Franz Carl Naegele (1778–1851), was proposed by Hermann Boerhaave, professor of medicine and botany at the University of Leyden (1709), and was merely quoted by Naegele.[4] Furthermore, the original latin text is ambiguous with respect to whether the calculation should be based on the first or last day of menstruation. Many women (if not most) deliver after their EDD calculated by Naegele's rule.[5] In 1782, Thomas Denman[6] noted "some inconveniences are produced by attempts to make exact reckonings for pregnant women: for when the time fixed for their delivery is past the mistake creates much solicitude and impatience. When therefore it is necessary to give an opinion on this subject it is better to mention some time beyond that which we really suppose or on the whole it would perhaps be better that labour should always come on unexpectedly".

200 years later this dilemma is just as evident. A given EDD leads most women to believe that the date is an accurate prediction of some considerable importance. It can be extremely difficult to allay the anxiety of a pregnant woman and her relatives if she remains undelivered 281 days or more after the date of her last menstrual period.



Proportion of patients undelivered by EDD in relation to maternal height.

Data derived from more than 23 000 consecutive pregnancies in which onset of labour occurred spontaneously.

Furthermore, it is likely that decisions about induction of labour are occasionally influenced by such worries. Even if prolonged pregnancy does carry an increased risk of perinatal mortality (and the data are by no means consistent), few would argue that this risk is substantial before 42 weeks' gestation in an otherwise uncomplicated pregnancy. The mean duration of pregnancy may be influenced by racial[7] and social factors. For example, in the UK North West Thames health region in 1988 the proportion of pregnant women undelivered by their EDD was strikingly influenced by maternal height (figure). Whatever the underlying mechanism, this example illustrates the fallacy of applying the present calculation to an entire population.

The alternative

In 1837 Montgomery[1] stated "a very common calculation among women themselves is to reckon 42 weeks from the last menstruation or 40 weeks from the middle day of the interval". If we were to follow that suggestion, and substitute this calculation for Naegele's rule, much anxiety

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would be alleviated and a potential stimulus for unnecessary obstetric intervention would be removed. An alternative solution would be to give women a range of dates (ie, from 38 to 42 weeks) during which they would probably go into labour, rather than a definite date. The final word should go to Boerhaave: he kept an elaborately bound volume, which was said to contain all the secrets of medicine. When it was opened after his death, all the pages were found to be blank—except one. Inscribed on this page was only the one sentence, which read, "Keep the head cool, the feet warm and the bowels open".

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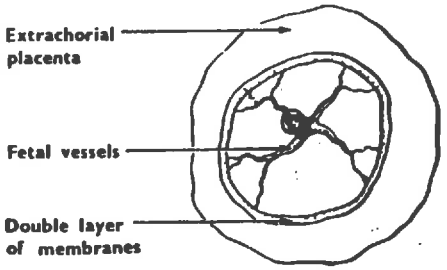


Fig. 3 Diagrammatic representation of an extra chorial placenta. A rim of placental tissue extends beyond the boundaries of the chorionic plate.

devoid of clinical significance and the circumvallate placenta is frequently associated with a rather small baby, and possibly with a slight excess of congenital malformations (Ladermacher et al, 1981). It is not accompanied by an increased perinatal mortality (Fox & Sen, 1972). Other aberrant forms of placentation are either of no functional significance, e.g. bilobate placenta, accessory lobes, or so rare that they can in practical terms be ignored, e.g. placenta membranacea, girdle placenta.

The site of cord insertion, (central, marginal or velamentous) is of no functional importance (Uyanwah-Akpom & Fox, 1977: Wood & Malan, 1978), though velamentous insertion does present a small risk because of the danger of trauma to the unprotected fetal vessels as they run through the membranes.

PLACENTAL WEIGHT

In routine practice it is virtually impossible to obtain a true estimate of placental mass after delivery, largely because of the variable quantities of maternal and fetal blood trapped within the placenta. Even if placental weight could be assessed accurately this information would, in itself, be of little value. Placental/fetal weight ratios are more meaningful but it would not be unfair to say that the only conclusion which can be drawn from the many studies of this ratio is that small babies usually have a small placenta and big babies have a big placenta. It then becomes almost an article of faith as to whether one believes that the baby is small because the placenta is small or that the placenta is small because the baby is small. If however fetal growth was strictly limited by placental mass then it would, of necessity, have to be assumed that during a normal pregnancy the placenta is at the full stretch of its physiological capacity with little or no functional reserve. Many observations suggest, however, that the placenta does have a considerable functional reserve. Thus, histopathological studies have shown that up to a third of the placental villi can be excluded from playing any role in materno-fetal transfer mechanisms, because they are entrapped in fibrin which fills in the intervillous space, without any effect on fetal growth or oxygenation (Fox, 1978). Further, experimental studies in sheep, involving either surgical reduction of placental mass (Robinson et al, 1979) or artificially increased fetal oxygen consumption (Lorijn & Longo, 1980), have confirmed the striking physiological reserve capacity of the placenta. It is therefore extremely unlikely that placental mass limits fetal weight and it thus appears that the placenta is small because the baby is small, the placenta sharing in the generally reduced growth of the fetal organs and its small size not acting as a contributory factor to fetal growth retardation.

PLACENTAL AGEING

There is a widely and tenaciously held view that the placenta ages as pregnancy progresses and that the term organ is on the verge of a decline into morphological and functional senescence. The villi of the term placenta are often described as showing all the morphological hallmarks of an aged tissue. However, this view is based almost entirely upon a misinterpretation of the structural changes which mark villous maturation and trophoblastic differentiation. These changes, unlike true ageing changes, increase the functional efficiency of the placenta. There are no morphological features of the term placenta that

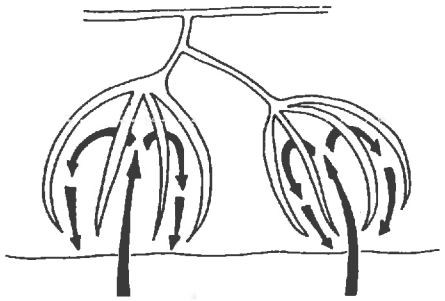


Fig. 1 Diagrammatic representation of two placental fetal lobules and of the maternal blood flow through these lobules

between the nuclei whilst microinjection studies show that substances flow freely through this layer and pass from villus to villus. This indicates that there is a continuous common cytoplasm over the entire surface of the placental villi (Gaunt & Ockleford, 1986). The syncytiotrophoblast is derived from the cytotrophoblastic cells, the syncytiotrophoblast being a terminally differentiated tissue and incapable of mitotic activity. The cytotrophoblastic cells are therefore the stem cells of the trophoblast but become less numerous and prominent as pregnancy progresses.

In the later stages of gestation the syncytiotrophoblast is focally thinned and anuclear. These attenuated areas usually overlie a dilated fetal capillary and appear almost to fuse with the vessel wall. These 'vasculo-syncytial membranes' represent the points of closest approximation of maternal and fetal blood and are specialised zones of the trophoblast for the facilitation of materno-fetal oxygen transfer.

The placental villi are bathed in maternal blood which fills the intervillous space. The blood enters the space from the uteroplacental vessels, flows slowly around the villi and then drains into basal venous outlets. It is important to note that the placenta depends solely on the maternal blood for its oxygen and nutrient supply.

DEVELOPMENTAL ABNORMALITIES

The only common developmental abnormality of the placenta is extrachorial placentation, in which the chorionic plate of the placenta, from which the villi arise, is smaller than the basal plate. The transition from villous tissue to non-villous membranes takes place therefore not at the placental margin but at some distance within the circumference of the fetal surface of the placenta (Fig. 3). If this transition is marked by a flattened ring of membranes the placenta is classed as 'circummarginate' whilst if this ring has a raised, rolled edge the placenta is 'circumvallate'. The clinical significance of extrachorial placentation has been much disputed but it is now clear that the circummarginate form is

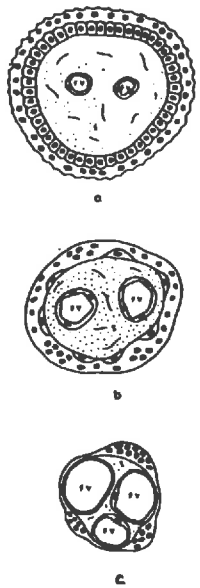


Fig. 2 Diagrammatic representation of the placental villi at various stages of pregnancy.
(a) A placental villus from a first trimester placenta.
(b) A placental villus from a second trimester placenta.
(c) A placental villus from a term placenta.
FV = fetal vessel, S = Syncytiotrophoblast

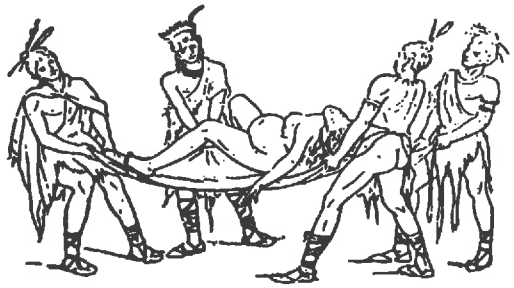
Ask The Midwife

By Ina May Gaskin

Q: What are some ways to help mothers who have trouble with back labor pain in their first labors?

A: I'd like to answer this question by writing about a recent birth I attended. The mother was eighteen and her partner twenty- two. Her water bag broke not long before midnight, and she was awake all night in light labor. If she had been in a hospital, her labor might have been described as "dysfunctional," since her cervix stayed at 2 cm of dilatation for many hours and was in such a posterior position that it could only be reached with the longest fingers (mine). The mother didn't like being checked, as she found any touching of her cervix painful. At the beginning of her labor, her cervix was totally effaced but not very thin (1 cm or so). Around midmorning, her labor began to increase in intensity, at least to the point that she began curling her toes with each rush. I showed her how to take a deep breath, expanding her belly as she did so, and how to very slowly let the breath out while keeping her mouth, jaw, bottom and legs relaxed.

She coped pretty well as long as she was making slow progress. Her cervix did get a little thinner over the long hours, but no other change was noticeable in it. By late afternoon there was another increase in intensity, and the mother could no longer be still. She tried walking outside, but cried and sobbed at how the downward pressure of the baby's head on her cervix caused great pain in her back. We let her know that it was all right to cry and encouraged her by saying that the pain was good ("bad" pain being the kind that signals damage to the body), since it meant that her cervix was trying to open.



When she got into a position from which cervical dilatation could be checked, we were all delighted to find that her cervix was very thin, in an anterior position and now dilated to 7 cm. Even though she was still in a lot of pain, the mother had to agree that this pain, at least in one sense, had been better than what had gone before. It was evident that this fast progress was going to continue. The only problem for all of us was the amount of pain the mother was experiencing in her back. She wasn't sure that she would be able to stand it much longer. We suggested that an all fours position would probably be the least uncomfortable and during a rush, her partner would push on her lower back. Still, she wanted more relief.

As I handled the backs of her thighs, I was struck by how unyielding her thigh muscles were. Dancers and horsewomen often have thighs like hers, and they just as often have some difficulty in relaxing through labor. Since I know how soft one has to get to really relax the muscles of the pelvic floor, I showed her partner how to shake her bottom during a rush, and as he did that, I shook one of her thighs in the same rhythm. I showed him how to avoid taking a "pinchy" grip and how to, instead, get hold of a big muscle group.

I have read several accounts of difficult labors among indigenous peoples in which a mother was shaken. Most of these accounts describe the shaking as if the caregivers were ignorant barbarians who were

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bent on hurting the mother. George Engelmann, for instance, wrote in 1882 in his **Labor Among Primitive Peoples**: "....I will briefly refer to some of those peculiar and barbarous methods to which these primitive people resort in their despair.....The Esthonians hold the patient in the air, shaking her vigorously if labor is retarded. In Syria, the patient is rolled in a blanket if she is not confined within twenty- four hours after the commencement of labor, and four male or female friends seize the corners of the blanket and roll the poor woman about in various directions, and bounce her up and down to facilitate confinement."

Dr. Palmer Findley in **The Story of Childbirth** (1933) mentions the same method, although with less of a judgmental tone: "Engelmann mentions a peculiar custom practised by some of our Western Indians in which the woman is tossed in a blanket, the four corners of which are held by four stout men, the idea being to correct any malposition of the baby and shake it out of the womb."

All of these accounts miss the point, I think. It's not that the baby is being shaken out of the womb, and shaking is not really a desperation measure. The real effect of shaking the mother in this way is pain relief and a considerable degree of relaxation. It may seem strange that a large body movement like shaking could accomplish this, but it works in the same way that rocking a baby works. It is hard to keep muscles contracted while being shaken rhythmically. It clears unnecessary tension from the muscles.

I learned this method of relaxation from my husband, and when I became a midwife, I found that shaking (some Chinese call it "chunging") worked beautifully with mothers who could not voluntarily relax the muscles of their legs and bottom. I was surprised and delighted to find out later that other peoples in widely separated parts of the world had made similar discoveries. It is not surprising that this method is found all over the world, since it demonstrates something true about the human nervous system, that uptightness in the muscles is like a stubborn opinion. When the body has to take advantage of its flexibility to compensate for being shaken, it can't remember the stubborn opinion.

I would be interested in hearing from anyone else who has found that shaking can be relaxing.

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Routine examination in the neonatal period

GD Moss, PHT Cartlidge, BD Speidel, and others. British Medical Journal, vol 302, no 6781, 13 April 1991, pp 878-879.

Objective — To assess the value of the second neonatal examination as a medical surveillance procedure.
Design — Prospective survey of routine neonatal examinations and the abnormalities identified during 8 March-30 June 1988.
Setting — Maternity unit with an annual birth rate of 5700.
Subjects — For first neonatal examination: 1795 babies born in the unit during the 115 day observation period. For second routine examination: 1747 babies (97.3%) discharged from postnatal ward.
Main outcome measures — Missed abnormalities (present but not previously noted); minor abnormalities (superficial infection or trivial or transient abnormalities not requiring intervention); and important abnormalities (unlikely to have been present at first examination but requiring intervention).
Results — An abnormality was detected in 158 (8.8%) infants on first neonatal examination. 1428 (79.6%) babies had a routine second examination, which disclosed 63 previously undetected abnormalities. Of these, seven (11%) would have been present on first examination, 49 (78%) were considered minor, and seven (11%) important — the most consequential being dislocatable hips (four infants). Thus an important finding was detected by only 0.5% of second examinations.
Conclusions — A second thorough examination in the early neonatal period cannot be justified as a screening procedure. A repeat examination of the hips alone in the first week of life is necessary.

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Midwifery

CLINICAL REVIEW

A contemporary view of the human placenta

H Fox

Our current knowledge of the human placenta is briefly reviewed. Particular stress is placed upon the considerable functional reserve capacity of the placenta, the unimportance of most visible abnormalities of the placenta, the lack of any evidence that the placenta ages during gestation and the lack of significance of placental weight. The effects on the placenta of infection and of maternal cigarette smoking are considered and the concept of placental insufficiency critically discussed. It is concluded that most cases of 'placental insufficiency' are, in reality, examples of maternal vascular insufficiency resulting from inadequate placentation during the early stages of pregnancy.

The placenta is, in functional terms, a highly complex organ though, without question, its most important task is to transfer oxygen and nutrients from the mother to the fetus. In a short review it is impossible to consider all aspects of the anatomy, physiology, endocrinology, immunology and pathology of the placenta and it is intended to discuss here only selected topics. Selectivity of this type can be invidious but the topics chosen are those which are considered to be of greatest practical importance to the midwife.

PLACENTAL STRUCTURE

Despite its many functional roles the placenta has a relatively simple structure. It is a villous haemochorial organ i.e. it has a villous structure and the chorion, or trophoblast, is in direct contact with maternal blood in the intervillous space. The organ consists of a number of subunits, or lobules, each of which resembles an inverted tree (Fig. 1). These lobules, it should be noted, do not correspond with the 'lobes' seen on the maternal surface whilst the term 'cotyledon' should be avoided when describing the non-cotyledonary human placenta.

Each lobule consists of a framework of stem villi which branch to form intermediate villi from which 'bud off' the terminal villi. The villi have a central mesenchymal core and a covering mantle of trophoblast (Fig. 2). The villous core contains mesenchymal tissue, macrophages (Hofbauer cells) and fetal capillaries which are initially small but dilate progressively throughout pregnancy until, at term, they occupy most of the cross sectional area of the villus.

In early pregnancy the villous throphoblast is of uniform thickness and consists of two layers of cells, an inner stratum of cytotrophoblastic cells with well defined limiting membranes and an outer layer of syncytiotrophoblast. The latter is a true syncytium for no cell boundaries are seen

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