obstetric trauma surgery
art and science

post fistula repair urine incontinence
step-by-step reconstruction
kees urethralization plus refixation

kees waaldijk
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obstetric trauma surgery
art and science
setting standards by evidence-based practice

post fistula repair urine incontinence
step-by-step reconstruction
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based on
findings and outcome
3,500 reconstructive incontinence procedures
2,350 patients
25,000 obstetric trauma procedures

the one and only risk factor of the obstetric fistula
poor obstetric care due to a failed system
all the rest is political blah blah blah rhetoric
deliberate lies and fake information

kees waaldijk
obstetric trauma surgery
art and science

series of textbooks each with a specific topic

setting evidence-based standards

this series has been developed for setting evidence-based standards in the training and management of the obstetric trauma in all its forms in the developing as well as in the industrialized world

the name of the series has been changed from obstetric fistula to obstetric trauma surgery since the fistula is only one aspect of the complex obstetric trauma

though a systematic approach is being followed this seems to be a utopia since the material is too extensive and it would take too long

each time a specific topic has been finalized it will be published as a separate entity; with later on an update if needed

then somewhere along the line a comprehensive summary will be produced in order to have a representative overview

the emphasis is placed on the functional anatomy of pelvis, pelvis floor and pelvis organ(s), the female urine and stool continence mechanisms, the mechanism of action and the principles of reconstructive and septic surgery

for training reasons it will follow a step-by-step approach and repetition; together with schematic drawings and photographs

the whole series is based on kees archives of obstetric trauma with so far 25,000 reconstructive and conservative procedures in 21,000 patients with a rare “complete” documentation of each procedure and results as to healing and continence by electronic reports with 150 parameters, over 100,000 pre/intra/postoperative digital photographs and a comprehensive database as personal experience over a 35-year period from 1984 up till now

as such it is considered to be a full scientific evidence-based report; though it has not followed the “you peer me, i peer you” doctrine

it is also not following the strict protocol of the international scientific journals or the so-called established theories; since only dead fish follow the flow of the river; and strict protocols kill any creativity; the message is not in the format

since it is the life work of the author it is written in his own words and in his own style

writing things down helps the author in organizing his own understanding and ideas
foreword

the surgical correction of genuine intrinsic stress incontinence, without anatomic tissue loss, is highly complicated and a multitude of theories have been postulated and even more operation techniques have been developed as based on tricks; however, all with mixed results since the real cause had not been solved and then addressed

so it is no surprise that the surgical correction of postrepair intrinsic stress incontinence, nb with anatomic tissue loss, is even more complicated with poor results

when the author started with the immediate management of the obstetric fistula he saw the real complex obstetric trauma with anatomic tissue loss

by analyzing these lesions he developed a scientific classification including postpartum incontinence which is also valid for postrepair incontinence

soon he realized that the obstetric fistula and urine intrinsic incontinence could only be solved by addressing the complex obstetric trauma as a whole whereby the fistula is closed and the continence restored by meticulous reconstruction of the functional pelvis anatomy ensuring physiology

based upon this principle he developed reconstructive operation technique principles for the different obstetric urine fistulas with excellent results

at a very early stage he understood that the pubocervical musculofascia (now he knows as anterior part of the endopelvic diaphragm) plays a major role especially with regards to continence

he started first to study the complicated female pelvis anatomy, then the topographic anatomy and as latest the functional pelvis anatomy in the female together with the continence mechanisms of the urinary, genital and digestive tract

then he developed another concept of the corpus endopelvinum = connective tissue body of pelvis with endopelvic diaphragm securing/stabilizing the pelvis organs in their variable anatomic position and as such supporting the urine/genital continence mechanism

having solved the theoretic problems and the practical consequences the author would like to share his experience and expertise as one textbook out of the series obstetric trauma surgery; art and science

this is the first time a scientific presentation is made of postrepair urine incontinence, its classification, its different real causes and the relevant reconstructive surgery principles for each type

the solution is not a trick but addresses the real cause

the author

October 2018
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prevention postrepair incontinence
prevention of postrepair incontinence
  at first or further repair

irrepairable fistulas/incontinence + ba hanya
irrepairable fistulas characteristics protocol
kees neovagina reconstruction
postdiversion urine incontinence

general discussion
postrepair incontinence
cause
management
prevention

basic science
mechanism of urine stress incontinence
endopelvic diaphragm
  another concept

postscriptum

abbreviations

measurements

references
endopelvic diaphragm
smooth muscle
intrinsic myogenic impulses
modulated by autonomic nervous system

perineum outlet diaphragm = pelvis floor
striated muscle
somatic innervation
one of the major problems in obstetric fistula surgery, besides closing the fistula, is the persistent postrepair intrinsic stress incontinence, fistula closed patient still leaking more or less continuously as if there still were a fistula

though it is already complicated to close the fistula at first or further attempt it is even more complicated to ensure continence even in very experienced hands

and still more complicated when persistent postrepair urine incontinence develops

though some kind of intrinsic stress urine incontinence is common immediately after fistula repair when the catheter has been removed there is a good chance of spontaneous total continence or social continence

this process can be promoted by a strict bladder drill, eventually combined with pelvis floor muscle exercises

however, if after 4-6 months no improvement has been obtained incontinence surgery may be indicated

the first priority is to exclude a residual (minute or larger) fistula as ectopic pacemaker as the cause of postrepair intrinsic stress incontinence which will heal by repair of this fistula; this was found in over 600 patients

if a residual fistula has been excluded one can proceed with incontinence surgery which constitutes

major reconstructive female pelvis surgery by reconstruction of all the defects within the endopelvic diaphragm and its fixation onto the anterobilateral pelvis wall

this book will outline a scientific classification as based on the characteristics of the original fistula, and the causes and the reconstructive surgery principles for each type

though we should aim at full continence, most patients are very happy once social continence has been achieved
postrepair urine incontinence
essentials

operation successful, patient still leaking

main problem
though closure of the fistula is possible in over 95% of the patients the occurrence of persisting postrepair urine incontinence longer than 6 months after repair is the largest major problem in obstetric fistula surgery which has not yet been fully solved so already at first attempt an effort should be made to restore continence as well

responsible factors
functional and/or anatomic tissue loss defects within the endopelvic diaphragm and its fixation onto the anterobilateral pelvis wall; see basic science

prevention (at first or subsequent repair)
paying meticulous attention to detail in reconstructing the functional pelvis anatomy by anatomic reconstruction of uv-junction, if necessary by end-to-end vesicourethrostomy, and repairing any defect in the pubocervical musculofascia/endopelvic diaphragm and its anterobilateral fixation onto pubis bones/arcus tendineus fasciae = atf avoiding all things which will lead to more scarring such as extensive dissection and all things which are not in line with the functional pelvis anatomy like wrong type of incision and wrong direction of closure against the natural tissue forces

visual confirmation
urine loss thru the external urethra opening = euo must be confirmed objectively by the surgeon with(out) coughing; if no urine loss thru euo there may still be a fistula

conservative management
some form of urine incontinence is frequent upon catheter removal and will disappear spontaneously within 2-3 months this process can be speeded up by bladder drill ie by continue to drink at least 4 liter per 24 hours and frequent micturition every 30-45 min; this will strengthen the physiologic process of bladder filling and micturition and train the continence mechanism if there is no relief within 3-4 months a surgical intervention should be planned, however by a surgeon with the necessary theoretical knowledge and practical skills

surgical management
since a minute fistula will act as pacemaker with objective urine loss thru euo with(out) coughing this has to be excluded by a dye test (up to 150 ml) under spinal anesthesia before the surgery is started; by closure of the minute fistula normally the incontinence will disappear if no fistula detected, identify the defect(s) responsible for the incontinence like urethra too short and/or too open and/or median or transverse defects in the pubocervical musculofascia as part of the endopelvic diaphragm or its fixation onto pubis bones/atf

irrepairable incontinence
still there remain patients with irreperable incontinence; see special chapter
principles incontinence surgery

physics of incontinence
once the intravesical excretory pressure becomes higher than the closing forces of the urethra there will be outflow of urine thru urethra either voluntarily as normal micturition or involuntarily and then it is called incontinence

cause of urine intrinsic stress incontinence
with endopelvic diaphragm as main supportive structure of the continence mechanism

- **aa** functional defects within the endopelvic diaphragm as in genuine urine intrinsic stress incontinence
- **bb** anatomic defects due to tissue loss of the endopelvic diaphragm with loss of its fixation to pelvis wall due to pressure necrosis as in postrepair urine incontinence
- **cc** combination of both aa and bb

principles of incontinence reconstructive surgery
meticulous repair of all the defects within the endopelvic diaphragm and its fixation onto the pelvis wall after exact identification of these defects

**genuine intrinsic stress incontinence** and also postrepair incontinence
meticulous repair of median longitudinal epd defect up to 2 cm to euo with proximal re-anchoring suture thru cervix  **kees urethralization**

**post IIAa incontinence**
kees urethralization plus meticulous repair of transverse/quartercircular epd defects with if necessary refixation onto paraurethra pubis bones

**post IIAb incontinence**
kees urethralization plus meticulous repair of quatercircular/semicircular epd defects with **anterobilateral** refixation onto lateral “atf”/inferior pubis bone/ischium bone periost and paraurethra pubis bone with closure of bilateral open paravesical space

**post IIBa incontinence**
kees urethralization plus meticulous **anterobilateral** epd re-fixation with also covering urehra onto intact paraurethra atf and onto paraurethra pubis bones

**post IIBb incontinence**
kees urethralization plus meticulous **anterobilateral** epd refxation onto lateral “atf”/inferior pubis bone/ischium bone periost and onto paraurethra pubis bones with closure of bilateral open paravesical space

**post I incontinence uncommon**
and then due to median ep defect and/or traction by fixed cervix or fibrosis

- **yy** longitudinal epd repair with re-anchoring cervix; see **genuine incontinence**
- **zz** bilateral epd fixation onto paraurethra pubis bones to neutralize traction by fibrosis and/or retracted/moving cervix/vault
functional pelvis anatomy
essentials

true pelvis cavity
a confined space for the distal outlet organs of the urinary tract anteriorly, the genital tract in the middle and the digestive tract posteriorly with hydrostatic and compression pressure; normally in a continent way and divided into

  anterior pre_subperitoneal compartment
for the distal end parts of the urinary tract: pelvic ureters, bladder and urethra

  median subperitoneal compartment
for the (also distal end parts of) genital tract: uterus, adnexa, cervix and vagina

  posterior retro_subperitoneal compartment
for the distal end parts of the digestive tract: rectum, anorectum and sphincter ani

enclosed by
  parietal pelvis fascia covering pelvis wall/floor muscles
and
  parietal peritoneum as boundary of intraperitoneal cavity
as connected to each other by
tela urogenitalis with corpus intrapelvinum and endopelvic diaphragm

corpus intrapelvinum as dynamic 3-dimensional matrix
connective tissue organ of pelvis consists of a cohesive mixture of collagen for strength, elastin for passive elasticity and plasticity and mostly smooth muscle fibers for dynamic active non-fatigue tonus in a loose, dense or condensed form as a dynamic matrix into which the organs and their supply are embedded and suspended/connected to the pelvis wall and each other by highly specialized structures protecting the organs and their supply against trauma and stabilizing/securing them in their variable anatomic position
as coordinated by intrinsic myogenic impulses and the autonomic nervous system considered to be a fluidum since no sharp demarcations between the archaic matrix and its specialized structures

endopelvic diaphragm
highly specialized structure of corpus intrapelvinum from symphysis anteriorly to sacrum posteriorly as connected to its bilateral arcus tendineus fasciae with cervix as centrum tendineum intrapelvinum since all musculofascia structures are connected to it as first line of counteracting intraabdominal hydrostatic pressure and supporting the urogenital continence mechanisms in their anatomic position and preventing herniation of the urinary tract, genital tract, intraperitoneal contents and distal digestive tract into the zero-pressure vagina

pelvis floor as one functional unit as part of abdominopelvic wall
levator ani muscles connected firmly to the perineum outlet diaphragm via perineal body and external sphincter ani muscle supporting and reinforcing each other levator ani muscles as “pelvis diaphragm” highly overrated with direct action on stool continence mechanism and only indirect action on urine continence mechanism perineum outlet diaphragm into which the end outlet organs with their striated sphincter mechanisms are anchored and supporting directly and the urine and stool continence mechanisms
female urine continence mechanism over in total 4-5 cm
bladder neck, uv-junction and whole urethra
supported by the endopelvic diaphragm and the perineum outlet diaphragm
there is an internal smooth muscle sphincter and an external striated rhabdosphincter
with washer effect by the mucosa and submucous vascular plexus
continence potential over its whole length

female genital continence mechanism over in total 3-4 cm
with cervix as internal smooth muscle sphincter as anchored into endopelvic diaphragm

female stool continence mechanism over in total 4-5 cm
anorectum and external sphincter ani
anchored within perineum outlet diaphragm
there is an internal smooth muscle sphincter and an external striated sphincter ani
muscle with washer effect by mucosa and submucous vascular plexus

urine stress incontinence mechanism genuine and post fistula repair
the anterior urethra wall is always fixed to the symphysis and cannot rotate backwards
away from the symphysis whilst the posterior urethra wall is mobile as supported by the
endopelvic diaphragm
once this support becomes weak the posterior urethra wall rotates backwards causing
funneling = vesicalization of the proximal and mid or whole urethra with a decrease in
outflow resistance so that the intrinsic closing forces can no longer counteract the intra
vesical expulsion forces
or by defects within the anchoring into perineum outlet diaphragm; isolated or combined

pelvis floor muscle exercises
have a positive effect upon the urine and stool continence mechanism since
the perineum outlet diaphragm contributes to the urine and stool continence mechanism
by further stabilizing the outlet parts
the levator ani muscles contribute directly to the stool continence mechanism to which
they are anatomically connected but only indirectly to the urine continence mechanism
with no anatomic connection whatsoever
with simultaneous reflex contraction of the external striated muscle sphincters
with increase in tonus of smooth muscle fibers of the endopelvic diaphragm by reflex
action via intrinsic myogenic impulses as modulated by the autonomic nervous system

obstetric trauma
due to hydrostatic pressure, dilatation of birth canal, (in)direct cutting thru, shearing and
compression; and in prolonged obstructed labor due to pressure necrosis
resulting in an enormous variety of defects from minimal to extensive

pelvis organ prolapse
herniation of adjacent high(er)-pressure organs into the zero-pressure vagina and then
further prolapse thru the vagina dragging vagina wall with them as intussusception
due to defects within the separating and supporting endopelvic diaphragm structures of
the corpus intrapelvinum between these organs and the vagina
levator ani muscles and perineum outlet diaphragm do not play a role in this process
since there is no anatomic contact between those organs and these structures

reconstructive surgery
the science is to identify the specific defects whilst the art is to reconstruct the functional
anatomy using the available dynamic autologous structures
endopelvic diaphragm trauma essentials

introduction

the endopelvic diaphragm as part of the corpus intrapelvinum or connective tissue body of the tela urogenitalis is an important dynamic structure as controlled by intrinsic myogenic impulses and the autonomic nervous system via a complex mechanism of reflex action (whereby baro- and stretch receptors play a major role)

securing the organs in their anatomic position and as such contributing to the different continence mechanisms

endopelvic diaphragm

from the pubis bone bodies anteriorly to the sacrum posteriorly and circumferentially connected to the pelvis wall like the skin of a drum or trampoline

it constitutes a real diaphragm with the cervix as its center with a small anterior median opening for the urethra and a larger posterior median opening for the rectum

and is divided into the following mainly smooth muscle components

pubovesical/posterior pubourethral ligaments (= muscles)
pubocervical musculofascia
arcus tendineus fasciae = atf
cervix
cardinal ligaments and broad ligaments
sacrouterine ligaments = rectouterinus muscles
rectovaginal fascia

mechanism of pathophysiologic action

due to downward intraabdominal pressure by the pregnant uterus the longitudinal fibers of the pubocervical musculofascia/endopelvic diaphragm may split in the midline

during childbirth itself the pressure changes from downward to upward; and semicircular compression and shearing occur at where the endopelvic diaphragm/pubocervical musculofascia is attached to pubis bones and atf

in prolonged obstructed labor pressure necrosis may develop and lead to anatomic tissue loss at any location within the endopelvic diaphragm

then there may be direct trauma (penetration, surgery) and trauma due to infection
defects within the endopelvic diaphragm

there are two types of defects viz defects without and defects with anatomic tissue

**aa** functional defects without anatomic tissue loss

**anterior defects**
with weakening of the urine continence support
by this mechanism genuine or postrepair intrinsic stress incontinence develops

**median longitudinal defects**
depending upon its location the posterior urethra, bladder base may herniate thru this
defect into the zero-pressure vagina and eventually prolapse to the outside

**central defect**
the cervix/uterus will herniate thru this defect into the vagina and then may prolapse
unopposed to the outside thru the hernia-prone opening in the pelvis floor dragging the
anterior vagina wall with it like intussusception

**apical defect**
this will result in herniation of the intraperitoneal contents into the zero-pressure vagina

**posterior defects**
this will result in herniation of the rectum into the zero-pressure vagina especially when
combined with perineal body defects

**lateral defects at atf**
this will result in loss of tonus of the endopelvic diaphragm resulting in an increase in the
caudad/cephalad movements but not in herniation/prolapse of an organ thru this defect

**lateral defects of the fascia sheath in between the atf and atlam**
this will result in medial displacement of the atf with loss of tonus and hypermobility of
the intrapelvic urogenital diaphragm but not in herniation/prolapse of an organ thru this
defect

**other location**
due to penetrating trauma or forceps delivery or vacuum delivery

**bb** defects with anatomic tissue loss

the extent and location of pressure necrosis lesions in prolonged obstructed labor may
be from minimal to extensive and from one location to the other in an endless variation
which makes the obstetric trauma so intriguing

**circular punched out defects**
the same size as the fistula or (slightly) bigger than the fistula

**transverse curved defects**
bigger than the fistula whereby the fistula is somewhere within this defect
defects within the endopelvic diaphragm
there are two types of defects viz defects without anatomic tissue
functional defects without anatomic tissue loss
anterior defects with weakening of the urine continence support
by this mechanism genuine or postpartum intrinsic stress incontinence develops
median longitudinal defects depending upon its location the posterior urethra, bladder base may herniate through this defect into the zero-pressure vagina and eventually prolapse to the outside
central defect the cervix/uterus will herniate through this defect into the vagina and then may prolapse unopposed to the outside through the hernia-prone opening in the pelvic floor dragging the anterior vagina wall with it like intussusception
apical defect this will result in herniation of the intraperitoneal contents into the zero-pressure vagina
posterior defects this will result in herniation of the rectum into the zero-pressure vagina especially when combined with perineal body defects
lateral defects at this will result in loss of tonus of the endopelvic diaphragm resulting in an increase in the caudad/cephalad movements but not in herniation/prolapse of an organ through this defect
lateral defects of the fascia sheath in between the atf and atlam this will result in medial displacement of the atf with loss of tonus and hypermobility of the intrapelvic urogenital diaphragm but not in herniation/prolapse through this defect
other location due to penetrating trauma or forceps delivery or vacuum delivery

defects with anatomic tissue loss
the extent and location of pressure necrosis lesions in prolonged obstructed labor may be from minimal to extensive and from one location to the other in an endless variation which makes the obstetric trauma so intriguing
circular punched-out defects the same size as the fistula or (slightly) bigger than the fistula
transverse curved defects bigger than the fistula whereby the fistula is somewhere within this defect
quartercircular defects with partial or total anatomic loss of atf and atlam and possible partial loss of levator ani muscles, obturator internus muscles and obturator membrane
with fistula formation and possible opening of the paravesical space
semicircular defects with partial or total anatomic tissue loss of atf and atlam; and with partial tissue loss of the levator ani muscles, obturator internus muscles and obturator membrane; eventually with bare bones
with fistula formation and opening of the paravesical space
(sub)total pubocervical musculofascia loss regularly (sub)total fascia loss with extensive fistula formation and anterior vagina wall loss and total loss of atf and atlam and (partial/extensive) loss of levator ani muscles, obturator internus muscles and obturator membranes is found with bare bones in a so-called empty pelvis with completely open paravesical space
(sub)total endopelvic diaphragm loss from time to time (sub)total loss of the whole diaphragm may be found with extensive soft tissue loss resulting in extensive urine/stool fistulas as cloaca for these unfortunate women nothing can be done surgically speaking

however, anatomic tissue loss may also be found
due to surgery whereby tissue is excised
or due to necrotizing infections like postmeasles noma vaginae

combination of functional with anatomic defects
this combination of aa and bb is always possible and has to be looked for

reconstructive surgery
it is important first to identify the real (extent of the) defect(s) and then reconstruct the functional anatomy meticulously using autologous structures so that normal physiology will be ensured whilst
special attention should be paid to make sure that the pubocervical musculofascia is properly (re)fixed onto the paraurethra pubis bones and bilateral atf and that the cervix is properly (re)anchored within the endopelvic diaphragm

discussion
the obstetric fistula is only part of the complex obstetric trauma, and only by repairing all defects in order to reconstruct the functional pelvis anatomy better postrepair results are ensured, especially as to postrepair continence
physiologic versus mutilating incision
anterior vagina wall

physiologic incision

mutilating incision
postrepair urine incontinence

classification

cause

postpartum urine incontinence
postrepair urine incontinence
operation successful, patient still leaking

as main problem

though closure of the fistula is possible in over 95% of the patients the occurrence of persisting postrepair urine incontinence longer than 6 months after repair is the largest major problem in obstetric urine fistula surgery

not only in the beginning but even in expert hands there is severe postrepair urine incontinence and postrepair post subsequent delivery incontinence

so already at first attempt an effort should be made to restore continence as well by meticulous repairing all the defects within the endopelvic diaphragm and its connection to the pelvis wall (pubis bones, atf); and anchoring of the cervix

responsible factors

functional and/or anatomic tissue loss defects within the endopelvic diaphragm and its fixation onto the anterobilateral pelvis wall; see basic science

poor understanding of functional pelvis anatomy

the problems at reconstructive pelvis surgery are poor understanding of the functional pelvis anatomy and the urine (in)continence mechanism, poor understanding of the enormous variety of the obstetric trauma and closing the fistula only without paying attention to the obstetric trauma as a whole and then as last poor surgical skills in reconstruction of the functional pelvis anatomy

the more the surgeon is familiar with the functional pelvis anatomy, the better he understands the urine continence and incontinence mechanisms, the better he is able to detect the enormous variety of lesions due to the obstetric trauma and the better his surgical skills, the better the outcome in terms of closure and continence

prevention (at first or subsequent repair)

paying meticulous attention to detail in reconstructing the functional pelvis anatomy by anatomic reconstruction of uv-junction, if necessary by end-to-end vesicourethrostomy, and repairing any defect within the endopelvic diaphragm and its fixation to its bilateral arcus tendineus fasciae = atf and paraurethra pubis bone

avoiding all things which will lead to more scarring such as extensive dissection and things which are not in line with the functional pelvis anatomy like wrong type of incision and wrong direction of closure against the natural tissue forces
objective visual confirmation

urine loss thru the external urethra opening = euo must be confirmed objectively by the surgeon with(out) coughing; if no urine loss thru euo there may still be a fistula

conservative management

some form of urine incontinence is frequent upon catheter removal and will disappear spontaneously within 2-3 months

this process can be speeded up by bladder drill ie by continue to dink at least 4 liter per 24 hours and frequent micturition every 30-45 min; this will strengthen the physiologic process of bladder filling and micturition and train the continence mechanism

and pelvis floor muscle exercises may play a positive role as well though this has never been objectively validated

if there is no relief within 3-4 months a surgical intervention should be planned, however by a surgeon with the necessary theoretical knowledge and practical skills

surgical management

since a minute fistula will act as pacemaker with objective urine loss thru euo with(out) coughing this has to be excluded by a dye test (by up to at least 150 ml) under spinal anesthesia before the surgery is started; by closure of the minute fistula normally the incontinence will disappear; this was found in 600 patients

if no fistula detected, identify the defect(s) responsible for the incontinence like urethra too short and/or too open and/or median or transverse or quartercircular or semicircular defects in the puocervical musculofascia as anterior part of the endopelvic diaphragm or its fixation onto atf

objective

though the aim should be total urine continence, this is not always possible; and social continence will be sufficient to re-socialize the woman back into her community

irrepairable incontinence

still there remain patients with unreparable incontinence; see special chapter

october 2018
postrepair urine incontinence is the major challenge in obstetric fistula surgery since most surgeons are only making an effort to close the fistula without paying attention to the obstetric trauma as a whole then there is incontinence after a subsequent delivery following successful repair with full continence

postrepair incontinence
a logical classification is according to the original fistula type

post kees I incontinence
post kees IIAa incontinence
post kees IIAb incontinence
post kees IIBa incontinence
post kees IIBb incontinence
post kees III incontinence

cause: various defects within the endopelvic diaphragm and other lesions like (sub)total loss of the anatomic continence mechanism and/or bladder prevention and incontinence surgery should aim at meticulous reconstruction of the functional pelvis anatomy by repairing all the defects within the endopelvic diaphragm if possible; either first at fistula repair or later on at incontinence surgery

postrepair postdelivery incontinence
also here a logical classification is according to the original fistula type

post kees I postdelivery incontinence
post kees IIAa postdelivery incontinence
post kees IIAb postdelivery incontinence
post kees IIBa postdelivery incontinence
post kees IIBb postdelivery incontinence
post kees III postdelivery incontinence

cause: new defects within the endopelvic diaphragm prevention only by good obstetric care incontinence surgery by first identifying and then meticulous repair of all the defects within the endopelvic diaphragm in order to reconstruct the functional pelvis anatomy if possible
cause postrepair incontinence

introduction
before one is undertaking measures to manage postrepair urine intrinsic incontinence one must know the mechanism of incontinence and the cause(s) in the different types

intrinsic stress incontinence mechanism
the anterior urethra wall is adherent to the posterior symphysis by loose connective tissue and a thin fluid film which allows the anterior urethra wall to shift against the symphysis friction free, though little; however it cannot rotate backwards away from the symphysis

the posterior urethra wall is firmly adherent to the pubocervical musculofascia with pubourethral ligaments as part of the endopelvic diaphragm

if defects develop within this diaphragm the posterior urethra wall rotates backwards away from the symphysis causing partial (or total) funneling of the proximal (or total) urethra since the anterior urethra wall stays sticking against the symphysis; this process is called vesicalization since functionally the funneled part of the urethra becomes part of the bladder (vesica)

besides backward rotation there is also backward shifting of the posterior urethra wall against the anterior urethra wall into the direction of the sacrum; since the anterior external opening is fixed and immobile

these two mechanisms of pathophysiologic action result into a wider (proximal) urethra lumen and a more oval elliptical arrangement of the smooth muscle fibers and

interfere with the intrinsic forces keeping the urethra closed since more force is needed to close the urethra; less resistance according to poiseuille law

once the intrinsic forces can no longer keep the urethra closed sufficiently this will lead to genuine or post fistula repair intrinsic stress incontinence in various degrees

in total intrinsic stress incontinence there is continuous leaking of urine on lying/sitting/standing/walking due to total loss of the intrinsic forces

intraoperatively under spinal anesthesia in these patients in the exaggerated lithotomy position the level of urine within the urethra is noticed in concord with respiration, rising on expiration and lowering on inspiration

a third mechanism may be a defect in the anchoring of the distal urethra (with external urethra opening) into the perineum outlet diaphragm; with or without avulsion

this mechanism is probably responsible for the development of postpartum genuine intrinsic stress urine incontinence with a hourglass or sandglass deformity of the urethra which is rather common; though combined with a median longitudinal defect within the endopelvic diaphragm
possible defects within endopelvic diaphragm

**all types of genuine and postrepair incontinence**

*ragged* median longitudinal defect with(out) dis-anchoring of anterior cervix

**post I incontinence**  
*not common*
most likely a median longitudinal defect with dis-anchoring of anterior cervix

**post II Aa incontinence**  
*additional defects*
transverse defects within the endopelvic diaphragm which may or may not be combined with indirect loose connection to the paraurethra pubis bones

**post II Ab incontinence**  
*additional defects*
still quarter- or semicircular defects within the endopelvic fascia combined with (in)direct loose connection to the paraurethra pubis bones and paraurethra atf
if no circumferential end-to-end vesicourethrostomy performed there may be a gap in the anterior uv-junction filled up by inert tissue

**post II Ba incontinence**  
*additional defects*
no or insufficient covering of urethra by endopelvic diaphragm with loose connection to paraurethra pubis bones

**post II Bb incontinence**  
*additional defects*
no or insufficient covering of urethra by endopelvic diaphragm with loose connection to paraurethra atf and/or paraurethra pubis bones
whilst the urethra may have been reconstructed from inert nonfunctional non-urethra tissue

**post III incontinence**
traction onto posterior urethra wall due to traction upon endopelvic diaphragm towards cervix/sacrum by scar tissue and/or median longitudinal defect with (partial) dis-anchoring of cervix as found in genuine incontinence and uterus/cervix prolapse

**tissue quality**
in all types of postrepair incontinence there is scar tissue in varying amounts depending upon the obstetric trauma and the kind of surgery; as due to the natural tissue forces
and it makes sense in surgery to classify the tissues as good, medium and poor in order to predict the outcome of the reconstructive surgery

**postrepair incontinence surgery**
depending upon systematic examination and identification of the cause a plan of action is devised and executed in order to reconstruct the **functional pelvis anatomy**
postpartum urine incontinence
obstetric trauma with(out) anatomic tissue loss

introduction

there is also urine leakage due to obstetric trauma however without tissue loss and the obstetric trauma surgeon has to be familiar with this

then there is objective stress/urge incontinence combined with a small/minute fistula

so the first priority is to identify the cause of the urine incontinence

and the second is to promote spontaneous healing by indwelling bladder catheter or if that is not successful by meticulous repair of the defects, if possible; whilst high oral fluid intake is a must

postpartum genuine intrinsic incontinence

as due to median defects within the endopelvic diaphragm or an overstretched loose endopelvic diaphragm so that the support of the posterior bladder/uv-junction/urethra is insufficient and these structures will rotate backward from the symphysis with funneling of proximal/mid or whole urethra and decrease in outflow resistance (equivalent to 4th power of radius of urethra diameter)

spontaneous healing is possible but the following is better
immediate catheter treatment for 4 weeks may promote spontaneous healing followed by pelvis floor exercises; if no cure obtained the responsible longitudinal median defect within the endopelvic diaphragm has to be repaired

postpartum hypotonic bladder with overflow incontinence

probably due to physical breakage/trauma of smooth muscle fibers of the detrusor muscle due to overstretching/filling of the bladder during delivery since urethra blocked by the infant head

spontaneous healing is possible via genuine intrinsic incontinence or intermittent self-catheterization; however the bladder remains full or is filling up again after each self-catheterization interfering with the physiologic healing processes

the following makes more sense
immediate bladder catheter for 4-6 weeks with complete decompression of the bladder at all times in order for the smooth muscle fibers to heal
uv-stricture with overflow incontinence and dysuria
	his may be due to scarring after subfistula trauma; and since it may be the cause or the sequel a minute fistula has to be excluded by a dye test

and needs (repeat) gradual dilatation with indwelling catheter for 2 weeks and high oral fluid intake

if no cure obtained a surgical revision of the uv-junction is needed with excision of all scar tissue and re-anastomosis of bladder neck to proximal urethra or buccal mucosa plasty

neurologic incontinence
	his is theoretically possible though difficult to diagnose and seems infrequent

obstetric fistulas combined with stress/urge incontinence

especially after fistula repair with minute/small residual fistulas, visible and nonvisible at direct inspection, whereby the scarring functions like ectopic pacemaker and prevents physiologic functioning of the continence mechanism

and a dye test is necessary to demonstrate or exclude the fistula

and a frequent finding as combined with ureter fistulas type III; either since this (scarring/proximal urine loss) functions as ectopic pacemaker or due to traction on the endopelvic diaphragm by the scarring

once the fistula has healed or the ureter has been successfully re-implanted, normally the incontinence will heal as well

postrepair post delivery incontinence

in patients who had a successful repair with full continence and who started to leak again without a fistula following a subsequent delivery; see next chapter

within the first 2 months after delivery the patients may benefit from indwelling catheter for 2-4 weeks and high oral fluid intake; however, most of them will not be cured

after a new or residual fistula has been excluded by a dye test incontinence surgery will be necessary whereby an effort has to be made to identify and then repair all the responsible defects within the endopelvic diaphragm and its suspension from/insertion into the pelvis wall
management
postrepair incontinence

chronology

step-by-step reconstruction
    kees urethralization plus refixation

post IIa incontinence
post IIb incontinence
post IIAa incontinence
post IIBa incontinence
post IIBb incontinence
post I incontinence  uncommon
genuine intrinsic stress incontinence
postrepair fibrosis/traction incontinence
discussion
chronology

development of incontinence surgery

though it is the end product which counts the development how to come to this is essential for a better understanding and insight

1983 start with obstetric trauma surgery

improving continence at first attempt closing the fistula

1984 bilateral fixation of bladder/epd onto symphysis; but the insight was missing
1984 physiologic incision and closure in line with natural tissue forces
1985 start with circumferential repair by end-to-end vesicourethrostomy in circumferential fistulas
1985 added to bilateral bladder fixation first by the martius fibrofatty pad graft which was used not to close the fistula but to elevate the bladder neck by suturing this graft over the repair onto the bilateral pubis bones/arcus tendineus fasciae and later even onto the backside of the anterior abdominal wall muscle fascia thru the paravesical space
1989 later on the author used the bilateral fixation of the anterior vagina wall (with adherent endopelvic diaphragm) onto the pubis bones after fistula closure to elevate the bladder neck
1985 start with early closure where the real obstetric trauma is still very visible
1992 transverse repair of epd defect in type IIa fistulas
1992 start with systematic immediate management and assessment of real obstetric trauma
1992 discontinuation of ff graft since it did not bring anything neither in closure nor in continence wherefore it was used; but concentrating upon repairing the obstetric trauma defects
1993 systematic measurement of bladder capacity by longitudinal diameter
1994 systematic measuring distance anterior bladder to anterior proximal urethra in circumferential fistulas in cm
1995 "large" lateral physiologic incision for lungu fistula for better access
1996 now intentionally direction of closure in line with natural tissue forces
2000-2005 developing insight into continence mechanism with concomitant attempt at reconstruction of functional pelvis anatomy
2005-now reconstruction functional pelvis anatomy with in process closure of fistula
postrepair incontinence

in 1984 adaptation of pubococcygeus muscles underneath bladder neck/uv-junction/proximal urethra

already in 1985 he started using strip(s) from the levator ani muscles by fixing these underneath the bladder neck bilaterally on the pubis bones for postrepair incontinence

if there was postrepair incontinence from 1987 he sutured the anterior vagina wall (he did not know then but now he knows + endopelvic diaphragm) bilaterally onto either the anterior abdominal wall musculature or if the paravesical space was scarred onto the pubis bones and/or obturator internus fascia as a broad sling, or better said hanging mat = hammock, around the urethra/bladder neck/bladder base; since a narrow sling would result in obstruction and cutting thru the tissues

distal lengthening urethroplasty up to anatomic euo mixed results; however, it is not the distal length but the proximal length which counts

kees urethralization by repair/rhaphy of pubocervical fascia underneath bladder neck, uv-junction, proximal urethra in order to lengthen proximal urethra (re-urethralization)

in 2001 a start was made with bilateral fixation of pubocervical musculafascia/avw onto paraurethra pubis bones/anterior abdominal musculature by 2x selaron each side at 3-4 and 4-5 cm from euo; with good results

in 2001 rhaphy of wide open distal urethra with bilateral fixation of mufascia/avw

in 2001 kees urethralization of bladder neck with bilateral mufascia/avw fixation

in 2001 kees urethralization + elevation by free fascia lata graft; good result but not further developed since mixed results with type IIba urethra reconstruction

in 2001 physiologic incision, kees urethralization by longitudinal rhaphy of fascia over bladder neck/proximal urethra whereby bilateral paravesical space opened with bilateral fixation of fascia onto atf/inferior pubis bones

from here on further development of technique whilst principles developed further

2002 repair of longitudinal defect within endopelvic diaphragm/pubocervical musculo fascia

2004 longitudinal urethra lengthening up to anatomic euo position by uy plasty; with mixed results

2005 start with refixation of epd/pcmuf onto paraurethra atf/pubis bones

2005-now further development of concept of endopelvic diaphragm with relevant reconstructive surgery principles

kees urethralization by longitudinal repair of endopelvic diaphragm with(out) cervix re-anchoring combined with anterobilateral epd (re)fixation
postrepair urine incontinence surgery
step-by-step kees urethralization plus refixation

introduction
the rationale of reconstructive surgery is to reconstruct the functional pelvis anatomy ensuring normal physiology

this seems already to be quite complicated in genuine incontinence without tissue loss considering the enormous number of techniques and principles

so even more in postrepair urine incontinence with variable amounts of tissue loss and variable amounts of additional surgical trauma like scar tissue

having solved the problems involved the author would like to present the principles in a step-by-step manner

history taking
when is the woman leaking: during daytime, at night, always and during which activity: whilst coughing, standing up, standing, walking, lying down, sleeping, sex

examination without anesthesia
the incontinence must be verified objectively with a filled bladder; in doubt suprapubic pressure push can be executed when she coughs

failed period of bladder drill + pelvis floor muscle exercises
a minimum of 3-4 months should be allowed before (repeat) surgical intervention since many women with postrepair incontinence are cured spontaneously by these methods and any surgical intervention is additional trauma

special surgical challenges
one is confronted with a short urethra with open deformed euo posteriorly drawn inside the vagina, scar and/or mutilation tissue and vagina strictures, fibrosis or shortening or even gynatresia in a patient as already operated by oneself or even worse by somebody else and everybody expects miracles

previous repair(s) varies from at least once up to over 10 times with repeat accumulated additional surgical trauma and scar tissue

one can hardly expect cooperation/compliance by the patient since at the first drop of urine after the incontinence surgery she will stop drinking

and how to ensure a normal functioning urine continence mechanism in this mess


step-by-step reconstruction
in line with the functional pelvis anatomy

this is divided into different parts
i  anesthesia, position, examination etc
ii  dye test
iii  physiologic incision + dissection
iv  identifying responsible defects epd/connection to pelvis wall
v  repair of longitudinal epd defect + if indicated re-anchoring cervix
vi  repair of any further defect within endopelvic diaphragm
vii  refixation of any loose epd connection to pelvis wall
viii  check of result + fixation of indwelling catheter
ix  adaptation of avw
x  urethra tissue quality
xi  documentation

i  anesthesia, position, examination etc

000
spinal anesthesia with long-acting agent

001
the patient is placed in the exaggerated lithotomy position with the legs flexed and slightly abducted in stirrups and her buttocks over the end of the operation table; this is the position of choice

002
a careful inspection and systematic examination (under anesthesia!) of the whole obstetric trauma is made like repair scar, distance euo to cervix or vault (euo/c) in cm, typing of incontinence if possible, position/state of euo etc

003
based upon this examination the postrepair incontinence is classified, and the surgeon makes up his definite plan of action how to handle this as its own unique entity

004
the labia minora are sutured onto the inside of the upper legs to keep the vagina open bilaterally
in order to improve the accessibility a uni- or bilateral episiotomy is performed at 4-5 and/or 7-8 o'clock or a small median episiotomy at 6 o'clock

then an auvard self-retaining weighted speculum is placed inside the vagina with underneath a gauze covering the anus to keep the vagina open posteriorly; no more specula.

**ii dye test**

**to exclude minute fistulas (ectopic pacemaker)**

a dye test is mandatory to exclude a minute fistula which may not be visible during the routine examination

this minute (or larger) fistula is responsible for the occurrence of urine intrinsic stress incontinence since it seems to function like an ectopic pacemaker

once the fistula has been repaired successfully, normally the incontinence will disappear as well

perform dye test with 50-100 ml asking patient to cough after instillation whilst keeping euo closed with catheter in situ and exert suprapubic pressure push

aa if positive by dye leakage and fistula identified, proceed further by closing this fistula according to the principles valid; nb this was found in over **600 patients**

bb if no dye but clear urine in vaginae this means a ureter fistula

c  if negative, no dye and no clear urine in vaginae, remove catheter and check for objective dye/urine thru euo which means real postrepair intrinsic-stress incontinence and proceed as following

at the same time check urethra length in cm and longitudinal bladder diameter in cm by euo/bw minus euo/b as starting reference point

**iii physiologic incision + dissection**

the surgical incision is an important part of any operation which should be chosen carefully in line with the natural tissue forces and executed carefully in order to obtain good access to the real operation field

a quarter- or semicircular physiologic incision is made at 2 cm proximally from euo thru or parallel to repair scar (if repair has been made by transverse incision) as within or parallel to ruga folds and also the same incision if repair has been made by longitudinal incision
in order to improve the accessibility a uni- or bilateral episiotomy is performed at 4–5 and/or 7–8 o'clock or a small median episiotomy at 6 o'clock then an auvard self-retaining weighted speculum is placed in the vagina with underneath a gauze covering the anus to keep the vagina open posteriorly; no more specula.

**Dye test to exclude minute fistulas (ectopic pacemaker)**

A dye test is mandatory to exclude a minute fistula which may not be visible during the routine examination this minute (or larger) fistula is responsible for the occurrence of urine intrinsic stress incontinence since it seems to function like an ectopic pacemaker once the fistula has been repaired successfully, normally the incontinence will disappear as well.

Perform dye test with 50–100 ml asking patient to cough after instillation whilst keeping uro closed with catheter in situ and exert suprapubic pressure push if positive by dye leakage and fistula identified, proceed further by closing this fistula according to the principles valid; nb this was found in over 600 patients if no dye but clear urine in vaginae this means a ureter fistula if negative, no dye and no clear urine in vaginae, remove catheter and check for objective dye/urine thru uro which means real postrepair intrinsic stress incontinence and proceed as following at the same time check urethra length in cm and longitudinal bladder diameter in cm by uro/bw minus uro/b as starting reference point.

physiologic incision + dissection

The surgical incision is an important part of any operation which should be chosen carefully in line with the natural tissue forces and executed carefully in order to obtain good access to the real operation field.

A quarter- or semicircular physiologic incision is made at 2 cm proximally from uro thru or parallel to repair scar (if repair has been made by transverse incision) as within or parallel to ruga folds and also the same incision if repair has been made by longitudinal incision.

the anterior vagina wall is dissected sharply from the endopelvic diaphragm using the scalpel and/or sharply curved thorek scissors in order to have a proper direct view of the endopelvic diaphragm and its connection to the pelvis wall.

**Systematic examination under spinal anesthesia + identifying the responsible defect(s)/factor(s)**

The better (all) the responsible factors are identified the better these can be corrected during the reconstructive surgery process and the better the chance of success; do not look for fascia tissue but look for defects within the shiny smooth muscle endopelvic diaphragm check for (loose) connection of endopelvic diaphragm to pelvis wall/cervix.

A meticulous examination is performed under spinal anesthesia as to uro deformed or not, wide or normal, in anatomic position or posteriorly drawn inside, urethra length, longitudinal bladder diameter, operation scars, tissue quality good, medium, poor.

Identify the responsible factor(s)

The possibilities are longitudinal, transverse, quartercircular, semicircular defects within the endopelvic diaphragm and/or direct or indirect loose connection of endopelvic diaphragm onto pubis bones/bilateral arcus tendineus fasciae with uni- or bilateral open paravesical space and.

Check for cervix anchoring within the endopelvic diaphragm.

kees urethralization by repair longitudinal defect; plus if indicated re-anchoring of cervix; this is essential in all types of genuine and postrepair incontinence.

The aim is to reconstruct the endopelvic diaphragm with cervix re-anchoring to ensure its dynamic function.

Repair the ragged median longitudinal defect by polyglycolic acid sutures with re-anchoring cervix with proximal suture.

So the posterior urethra wall, posterior uv-junction and posterior bladder are prevented from herniating into the vagina.
repair the transverse, quarter- or semicircular defects within the endopelvic diaphragm by refixation of endopelvic diaphragm to paraurethra pubis bones periost and paraurethra atf over these defects.

the aim is to reconstruct the endopelvic diaphragm and its connection to the pelvis wall to ensure its dynamic function.

014
refix the lateral endopelvic diaphragm onto bilateral paraurethra atf by 1-2x polyglycolic acid sutures each side.

check for continence and measure urethra length in cm.

015 important to neutralize the traction towards the cervix/sacrum. make small deep transverse paraurethra incisions up to pubis bone periost otherwise fixation may slip during the immediate postoperative period or later and then refix paramedian endopelvic diaphragm onto paraurethra pubis bone periost by 2x polyglycolic acid suture each side.

this is important since it will prevent retraction/shifting of the endopelvic diaphragm + adherent posterior urethra wall towards the sacrum.

016
if done correctly there will be normalization of euo with reduction of posterior euo into anatomic position since traction onto endopelvic diaphragm towards cervix/sacrum has been neutralized; and increase in urethra length.

vii refix any quarter- and semicircular loose connection.
only if an intact endopelvic diaphragm is circumferentially connected to the pelvis wall can an increase in its tonus produce cephalad/anterior elevation.

viii tissue handling, sutures etc.
tissue handling is an important part + suture technique has to be perfected at each operation.

017
good bites are taken to get broad adaptation of the raw endopelvic smooth muscle onto pubis bone periost and arcus tendineus fasciae.

018
care is taken to apply sufficient tension in order to counteract the traction towards the cervix/sacrum in order to neutralize this traction.
019
care is taken only to pick up the smooth muscle endopelvic diaphragm and on picking up the atf also the “underlying” pubis bone periost

020
cave do not cut the sutures too short since then the knot(s) will slip and loosen up

ix check for continence after each step

by checking for continence after each step the surgeon will notice at which stage continence becomes evident; to improve his own insight

021
with urine or normal saline inside the bladder ask patient to cough (+ suprapubic pressure push onto anterior abdominal wall) and look if there is urine leakage thru euo

x final check + fixation of catheter

final check with measurements with documentation for transparent audit and indwelling bladder catheter for 2 weeks for total bladder decompression promoting the healing of the endopelvic diaphragm

022
check for continence and measure urethra length/longitudinal bladder diameter in cm by measuring the distance euo to bladder wall (euo/bw) by calibrated metal sound, then insert foley catheter and determine urethra length by measuring distance euo to balloon (euo/b)
euo/bw minus euo/b = longitudinal bladder diameter (in cm)

023
nb normally the final urethra length at operation ending is a minimum of 1.5-2 cm and at least 1 cm longer then the initial urethra length at operation beginning resulting in normal-width good/medium/poor-quality urethra_euo in anatomic position

024
insert nelaton ch 16, fix it and check for urine drainage thru catheter
if urine is draining this means 3 things:
a the catheter is inside bladder
b at least one ureter is draining into the bladder and
c the patient is not in shock
if no draining of urine, check for the cause and correct it

indwelling bladder catheter choice
the author prefers a nelaton catheter since it has a big bore; so better drainage than foley catheter with small bore; if foley catheter is used do not balloon but fix it
anterior vagina wall adaptation, episiotomy etc

since the vagina is never sterile the anterior vagina wall is only adapted by a couple of everting sutures to allow free evacuation of blood clots, tissue debris and bacteria in line with septic surgery principles

025
the anterior vagina wall is only adapted quartercircularly by 2-4 everting absorbable or nonabsorbable sutures

026
if episiotomies have been performed these are adapted

027 optional
the vagina is packed tightly with gauze (soaked in antiseptic or not) to help hemostasis though normally complete hemostasis is secured

028 cave
if there is no urine flow, not even after attempts at forced diuresis, this is an indication that both ureters have been traumatized and the whole repair has to be undone

029
if the patient is in good condition with good urine flow she is transferred to the post operative ward.

tissue quality

during the operation procedure the urethra tissue has to be classified as good, medium or poor in order to predict the outcome and to evaluate the results; this has to be entered into the operation report as documentation

documentation

since documentation is an important part of any type of surgery, analysis of technique, transparent audit and scientific process

an operation report has to be written in detail including complications; with prediction of healing and continence on a 5% scale from 5% to 95%; immediately after the surgery however, by writing an operation report the surgeon’s action becomes fully transparent and open to criticism by others, especially by the verbal "surgeons" who have to prove their value by being vocal

then the outcome of the surgery has to be documented as well against all the parameters to assess if one is on the right track or not and then continue and refine the technique or take the necessary action
special attention in the different types

post type I incontinence uncommon
proximal median longitudinal splitting of endopelvic diaphragm fibers with bilateral retraction and cervix dis-anchoring

though the continence mechanism is not involved in a very small portion incontinence may develop due to functional lesions within the endopelvic diaphragm

longitudinal repair of bilaterally retracted endopelvic diaphragm with re-anchoring of cervix is indicated

the same as in genuine intrinsic-stress incontinence

prognosis good in over 95%

post type IIa incontinence
distal transverse defects within the endopelvic diaphragm with(out) indirect loose connection to paraurethra atf and paraurethra pubis bone

transverse repair of the endopelvic diaphragm is indicated which will correct the indirect loose connection as well

other defects within the endopelvic diaphragm have to be looked for and if present have to be repaired

prognosis good in 90-95%

post type IIb incontinence
quarter- to semicircular circular defects within the endopelvic diaphragm with partial/total loss of atf/atlam, partial loss of lam, no quarter- or semicircular antero (bi)lateral connection to pelvis wall with (bi)lateral open paravesical space (eventually bare pubis/ischium bones)

quarter- to semicircular repair of endopelvic diaphragm by anterobilateral refixation onto paraurethra atf/pubis bone periost and paraurethra pubis bone periost

however, it may be that one side is (far) more affected than the other; so a customized approach is necessary

prognosis good in 80-85%
post type IIb incontinence
no covering of urethra by endopelvic diaphragm with loose connection to paraurethra atf and paraurethra pubis bone periost

anterobilateral refixation of endopelvic diaphragm over urethra onto paraurethra atf and paraurethra pubis bone is indicated

good prognosis in 85-90%

post type IIb incontinence
no covering of urethra by endopelvic diaphragm, partial or total loss of atf/atlam, partial loss of lam, partial loss of obturator internus muscle and semicircular loose connection to anterolateral pelvis wall with bilateral open paravesical space and bare pubis/ischium bones often combined with severe traction towards sacrum/cervix due to (extensive) fibrosis/mutilation by obstetric trauma and/or surgery

semicircular refixation of endopelvic diaphragm onto bilateral atf and over urethra onto paraurethra atf and paraurethra pubis bone periost is indicated; however, this is very complicated

good prognosis so far in some 60% but improving due to better understanding and refining of technique though the majority are inoperable

severe fibrosis/mutilation with traction towards sacrum due to the obstetric trauma, due to cesarean section with paradoxic moving on cough, but mostly due to (repeat) surgery by incompetent surgeons though it happens as well in experienced hands

this is a very serious problem since nobody is willing to address it since the aim is quantity instead of quality

if still operable an effort has to be made to neutralize the traction by anterobilateral fixation of avw_cervix_endopelvic diaphragm onto pelvis wall under high tension

with special attention on anterior epd pcmuf fixation onto paraurethra pubis bone periost in order to neutralize the traction towards the sacrum

good prognosis in only 10-20% though the great majority are inoperable
discussion

these reconstructive surgery principles are the end-product of a 35-yr-long extensive and intensive innovative theoretic, clinical and surgical research into the obstetric trauma and the functional pelvis anatomy and urine (in)continence mechanism

which is still in process since it keeps on fascinating the author

since the fresh lesions of the natural experiment of the complex obstetric trauma are the clue to a better understanding than all the postmortem examinations (very necessary for some basic facts) and indirect imaging (vague/difficult to interpret) with their resulting theories and operation techniques

this is the first time a systematic step-by-step approach to postrepair incontinence has been described in a scientific way as based on

the functional pelvis anatomy
the urine (in)continence mechanism
the causative factors/defects as identified
postsurgery evidence-based outcome

with full explanation of the incontinence mechanism and the causative factors involved

with the endopelvic diaphragm as another concept for securing the pelvis organs in their variable anatomic position and as such supporting the continence mechanisms of the different tracts

the most important is the longitudinal repair of the ragged median longitudinal defect within the endopelvic diaphragm with bilateral (re)fixation onto the paraurethra atf and paraurethra pubis bones

kees urethralization plus refixation

by this the author thinks he found the causal solution of genuine and of postrepair urine stress incontinence in a physiologic way by anatomic correct reconstruction of the functional pelvis anatomy

in genuine intrinsic urine incontinence the evidence-based success rate is over 95% in the 398 consecutive patients operated so far
physiologic versus mutilating incision
anterio r vagina wall

physiologic incision

mutilating incision
step-by-step epd reconstruction

ragged median epd defect

plus defective fixation onto paraurethra atf/pubis bone
step-by-step epd reconstruction

first epd suture re-anchoring cervix

second epd suture

third epd suture

fourth epd suture

fifth epd suture

median epd defect reconstructed

© kees
step-by-step epd reconstruction

defective fixation onto paraurethra atf/pubis bone

refixation 1-2 x onto R paraurethra atf

refixation 1-2 x onto L paraurethra atf

refixation 1-2 x onto paraurethra pubis bone

refixation completed

epd + fixation reconstructed

© kees
post kees IIa incontinence  
+  
post kees IIa postdelivery incontinence

type kees IIa fistulas  
in **type IIa** fistulas there is tissue loss of the bladder, urethrovesical junction/trigonal ring, detrusor loops, proximal-(mid) urethra, endopelvic diaphragm/pubocervical musculofoasia and anterior vagina wall (and cervix and/or uterus) with **minor to moderate** involvement of the continence/closing mechanism

there may be slight trauma to the arcus tendineus fasciae (**atf**), arcus tendineus of the levator ani muscle (**atlam**) and levator ani musculature

there may be a transverse/quartercircular defect in the endopelvic diaphragm/pubocervical musculofoasia so that its connection onto the paraurethra pubis bones and paraurethra atf is **indirectly** disrupted or weakened but the atf itself is more or less intact; and so is the atlam

**post kees IIa incontinence**  
in **post IIa** and **post IIa delivery incontinence** the fistula has been closed/healed but there is a remaining transverse or quartercicular defect within the endopelvic diaphragm with **indirect loose** anterolateral fixation to pelvis wall

since the **posterior** bladder neck, posterior uv-junction and posterior urethra wall are not supported properly these structures will rotate backwards away from the symphysis resulting in funneling of the proximal urethra with a decrease in outflow resistance and intrinsic stress incontinence

there may be also **traction towards the cervix/sacrum** with shifting of the posterior urethra wall in respect to the anterior urethra wall resulting in posterior euo deformation and distortion of the urethra architecture also with a decrease in outflow resistance and intrinsic stress incontinence

**possible epd/pcmuf defects** besides ragged median defect

- **aa** defects slightly bigger than original fistula
- **bb** transverse distal defects
- **cc** quartercircular defects
- **dd** indirect loose connection onto paraurethra pubis bones

however **intact atf/atlam** and **closed paravesical space**

resulting into **traction onto posterior urethra wall towards cervix/sacrum** by endopelvic diaphragm with distortion of urethra architecture  
this traction may be reinforced by eventual scarring/fibrosis of avw/epd either due to the obstetric trauma or due to (repeat) surgery  
with concomitant proximal longitudinal defect with(out) dis-anchoring of cervix
incontinence surgery
reconstruction endopelvic diaphragm/pubocervical musculofascia
the responsible defects within the endopelvic diaphragm are repaired meticulously by a single layer of interrupted polyglycolic acid sutures; with if necessary bilateral fixation of the endopelvic diaphragm to the paraurethra pubis bones by 2 polyglycolic acid sutures each side to neutralize the traction towards the cervix/sacrum resulting in normalization of the urethra width and euo into anatomic position

step-by-step reconstruction
in line with the functional pelvis anatomy
this is divided into different parts
i anesthesia, position, examination etc
ii dye test
iii incision + dissection

special attention post IIa (postdelivery) incontinence
iv identifying responsible defects epd/connection to pelvis wall
transverse or quartercircular defect with loose indirect connection to anterobilateral pelvis
v repair of longitudinal epd defect + re-anchoring cervix
longitudinal repair of ragged median epd defect plus if indicated re-anchoring anterior cervix
vi repair of any defect within endopelvic diaphragm
meticulous transverse/quartercircular repair of the epd defect with single layer of interrupted polyglycolic acid sutures
vii refixation of any loose epd connection to pelvis wall
if necessary (open euo and/or still urine thru euo on cough/pressure) bilateral fixation of epd onto paraurethra pubis bone periost by 2x polyglycolic acid sutures each side
viii check of result + fixation of indwelling catheter
ix adaptation of avw
x urethra tissue quality
xi documentation
type kees IIa fistulas
post IIa incontinence

fixation epd onto paraurethra pubis bone

fixation epd onto paraurethra pubis bone
post IIa incontinence

fixation epd onto paraurethra pubis bone

fixation epd onto paraurethra pubis bone
post kees IIAb incontinence
+
post kees IIAb postdelivery incontinence

type kees IIAb fistulas
in type IIAb fistulas there is circumferential tissue loss of bladder neck, urethrovessical junction/trigonal ring, detrusor loops, proximal-(mid) urethra and tissue loss of posterior pubourethral ligaments, pubocervical musculofascia, anterior vagina wall (and cervix and/or uterus), atf, atlam, pubococcygeus muscles and obturatococcygeus muscles; also there may be even trauma to the obturator internus muscles, obturator membrane and coccygeus muscles (bare pubis/ischium bones) with eventual loss of the pubis bone periost and the pubis symphysis cartilage with moderate to major involvement of the continence/closing mechanism

there is no functional tissue connection whatsoever between the traumatized urethra or what is left of it and the traumatized bladder (neck) whereby the urethra has retracted distally and the bladder proximally in opposite directions

this distal and proximal retraction is limited by the fact that the anterior urethra is loosely fixed/adherent to the posterior symphysis and that the anterior bladder is loosely fixed adherent to the posterior symphysis and anterior abdominal wall; however, sometimes the bladder has slipped above the cephalad brim of the pubis symphysis

the stabilizing support of the endopelvic diaphragm needed for the physiologic urethra continence/closing function has been lost since there is no connection whatsoever between what is left of the traumatized pubocervical musculofascia and the paraurethra pubis bones since a quarter- or semicircular part of the endopelvic diaphragm/pubocervical musculofascia is missing

for a good understanding it is the cephalad part of the levator ani muscle together with its origin atlam which is lost whilst the caudad part with its insertion into the levator plate and coccyx is still intact

if there is major loss of the levator ani musculature the result is an empty pelvis with bare pubis bones; this is always combined with (sub)total loss of the atf and atlam from paraurethrally up to the ischium spine

the distal paraurethra atf/atlam and pubis bone insertion of the endopelvic diaphragm is intact whilst the proximal insertion of atf/atlam into ischium spine and median insertion into paraurethra atf/atlam are lost with bilateral open paravesical space

post kees IIAb incontinence
in post IIAb and post IIAb postdelivery incontinence there may be still quartercircular or semicircular defects within the endopelvic diaphragm with partial or (sub)total loss of atf and atlam with (in)direct loose connection onto paraurethra pubis bones and paraurethra atf
possible epd/pcmuf defects besides ragged median defect

aa quartercircular defects with
bb semicircular defects
cc loss of atf/atlam over proximal/middle third over 4-5-6 cm with distal 1-2-3 cm intact;
(direct) loose connection to anterobilateral pelvis wall
dd bilateral open paravesical space

possible pelvis muscle loss
partial tissue loss of levator ani muscle, obturator internus muscle, obturator membrane,
(ischio)coccygeus muscle, piriormis muscle
with
bare pubis/ischium bones

with proximal longitudinal pcmuf/epd defect with dis-anchoring of cervix

step-by-step reconstruction
in line with the functional pelvis anatomy
this is divided into different parts
  i  anesthesia, position, examination etc
  ii  dye test

specific attention post IIAb (delivery) incontinence

  iii  incision and dissection
physiologic inverted U incision at 1.5-2 cm from euo within or parallel to ruga folds thru bilateral deep sulci (up to superior pubis bone/ilium bone); if possible thru repair scar and dissection of what is left of avw from what is left of pcmuf/epd

  iv  identifying responsible defects/connection to pelvis wall
(up to large) anterior pcmuf/epd loss and partial direct loose connection onto bilateral pelvis wall (with partial/large loss of atf) and indirect loose anterior connection (paraurethra pubis bone body)

  v  repair of longitudinal epd defect + if indicated re-anchoring cervix
longitudinal repair of ragged median epd defect plus if indicated re-anchoring anterior cervix
**vi + vii** repair/refixation of any pcmuf/epd defect

**aa** start with **freshening** paraurethra pubis bone body and bilateral refixation of pcmuf/epd onto paraurethra pubis bone periost by 2x polyglycolic acid sutures each side to restore the anterior connection of the epd and to prevent slipping of this connection and to neutralize traction onto the posterior urethra wall by retraction of epd towards the sacrum.

**bb** then bilateral refixation of pcmuf/epd at 10-11 hr at anterolateral atf/inferior pubis bone periost by 1x polyglycolic acid suture each side.

**cc** if indicated with bilateral refixation of pcmuf/epd at 9-10 hr onto lateral “atf”/ischium bone periost/obturator internus muscle fascia by 1x glycolic acid suture each side; if possible at cervix level.

**nb** now the bilateral open paravesical space has been closed by the semicircular “anatomic” reduction/repositioning/refixation of the endopelvic diaphragm onto the anterobilateral pelvis wall with normalization/repositioning of euo into its anatomic position.

**viii** check of result + fixation of indwelling catheter

**ix** adaptation of avw

**x** urethra tissue quality

**xi** documentation
type kees llAb fistulas
post IIAb incontinence

fascia defect type IIAb fistula
atf/atlam loss

quartercircular trauma

semicircular trauma

semicircular trauma

© kees
post IIAb incontinence

fixation epd onto “atf”/pubis bone

© kees
post IIAb incontinence

fixation epd onto paraurethra pubis bone

fixation epd onto paraurethra pubis bone
post IIAb incontinence

fixation epd onto paraurethra pubis bone
post kees II Ba incontinence
+
post kees II Ba postdelivery incontinence

type kees II Ba fistulas
in type II Ba fistulas there is major tissue loss of the urethra and tissue loss of the urethrovessical junction/trigonal ring, detrusor loops, bladder, pubocervical musculosfascia and anterior vagina wall (and cervix and/or uterus) with major involvement of the continence/closing mechanism; though there is tissue loss of the anterior part of the pubocervical musculosfascia the atf, atlam and levator ani musculature are intact though the connection of the musculofascia/epd onto the immediate paraurethra pubis bones may be lost

type kees II Ba incontinence
in post II Ba and post II Ba postdelivery incontinence the urethra reconstruction has been successful but the urethra is not sufficiently covered/supported by the endopelvic diaphragm with loose indirect anterobilateral connection to the pelvis wall and direct loose connection to paraurethra pubis bones whilst the paravesical space is closed

possible pcmuf/epd defects besides ragged median defect
aa distal epd/pcmuf defect
bb direct loose connection onto paraurethra atf and paraurethra pubis bones however intact atf/atlam and closed paravesical space

resulting into traction onto posterior urethra wall towards cervix/sacrum by endopelvic diaphragm with distortion of urethra architecture

with or without longitudinal proximal pcmuf/epd defect with dis-anchoring of cervix

step-by-step reconstruction
in line with the functional pelvis anatomy

this is divided into different parts
i anesthesita, position, examination etc
ii dye test

special attention post II Ba (delivery) incontinence

iii incision + dissection
physiologic inverted U incision at 1 cm from euo parallel to ruga folds and sharp wide dissection of avw from epd/pcmuf
iv identifying responsible defects epd/connection to pelvis wall
neourethra not covered sufficiently by epd/pcmuf
loose connection to pubis bone and intact paraurethra atf

v repair of longitudinal epd defect + re-anchoring cervix
meticulous longitudinal repair of ragged median epd defect plus if indicated re-anchoring anterior cervix

vi + vii repair/refixation of any pcmuf/epd defect

vii 0a bilateral refixation of pcmuf/epd at 10-11 hr at anterolateral intact atf/inferior pubis bone periost by 1x polyglycolic acid suture each side

vii 0b freshening paraurethra pubis bone body and bilateral refixation of pcmuf/epd onto pubis bone periost by 2x polyglycolic acid sutures each side to restore the anterior connection of the epd and to prevent slipping of this connection

vi 0c whereby at the same time the neourethra will be covered by epd

viii check of result + fixation of indwelling catheter
ix adaptation of avw
x urethra tissue quality
xi documentation
identifying responsible defects: epd/connection to pelvis wall
neourethra not covered sufficiently by epd/pcmuf
lesion connection to pubis bone and intact paraurethra.

Repair of longitudinal epd defect plus if indicated re-anchoring anterior cervix.

Repair/refixation of any pcmuf/epd defect.

Bilateral refixation of pcmuf/epd at 10-11 hr at anterolateral intact atf/inferior pubis bone periost by 1x polyglycolic acid suture each side.

Freshening paraurethra/pubis bone body and bilateral refixation of pcmuf/epd onto pubis bone periost by 2x polyglycolic acid sutures each side to restore the anterior connection of the epd and to prevent slipping of this connection.

Whereby at the same time the neourethra will be covered by epd.

Check of result + fixation of indwelling catheter.

Adaptation of avw/urethra tissue quality.

Documentation.
step-by-step epd reconstruction post llBa

fixation epd onto paraurethra atf/pubis bone

fixation epd onto paraurethra atf/pubis bone
step-by-step epd reconstruction post II Ba

fixation epd onto paraurethra atf/pubis bone

fixation epd onto paraurethra atf/pubis bone
post kees IIb incontinence + post kees IIb postdelivery incontinence

type kees IIb fistulas
in type IIb fistulas there is (sub)total circumferential tissue loss of the urethra, urethro-vesical junction/trigonal ring, bladder neck, detrusor loops and tissue loss of the intermediate and posterior pubourethral ligaments, pubocervical musculofascia, anterior vagina wall (and cervix and/or uterus), atf, atlam, pubococcygeus/obturatococcygeus muscles; also there may be even trauma to the obturator internus muscles, obturator membrane and coccygeus muscles (bare pubis/ischium bones) with eventual loss of pubis bone periost and pubis symphysis cartilage with extensive involvement of the continence/closing mechanism.

there is no functional tissue connection whatsoever between what is left of the severely traumatized urethra if anything is left at all and the bladder (neck) whilst the bladder has retracted proximally which is limited anteriorly by the loose fixation of the anterior bladder onto the posterior symphysis and anterior abdominal wall

the quarter- or semicircular defect within pubocervical musculofascia is separating the proximal part of the diaphragm completely from the distal part together with bilateral loss of the distal, mid and eventually proximal atf from paraurethrally up to the ischium spine

the cephalad part of the pubococcygeus muscle has been lost with (sub)total loss of the paraurethra atlam; in extensive trauma also the cephalad part of the obturatococcygeus muscle is lost with complete atlam loss from paraurethrally up to the ischium spine

normally there is very extensive tissue loss which makes the surgical management so complicated in these fistulas; frequently an empty pelvis is found with bare pubis bones and sometimes the fistula may be inoperable right from the beginning

these fistulas are characterized by major or (sub)total pubocervical musculofascia/endopelvic diaphragm loss with(sub)total loss of avw, (sub)total loss of atf and atlam and completely open bilateral paravesical space and major loss of the anatomic urine continence mechanism

and have the lowest chance of healing and continence; many present right from the beginning as “inoperable” or really inoperable

post type kees IIb incontinence
in post IIb and post IIb postdelivery incontinence the amount of anatomic tissue loss is enormous which makes it very complicated to do something

the two anterior vagina wall sulci are very deep up to the superior rami of the pubis bones and up to the ilium bones with bilateral open paravesical space
still some things can be done to reinforce the continence mechanism by meticulous repair of what is left of the endopelvic diaphragm

still do not expect too much from the incontinence operation considering the enormous tissue loss in these fistulas

**possible epd/pcmuf defects** besides the ragged median defect

- **aa** distal or (sub)total pcmuf/epd loss
- **bb** major (sub)total loss atf/atlam over 7-7.5 cm with
- **cc** complete loose anterobilateral connection to pelvis wall
- **dd** bilateral open paravesical space

**possible pelvis muscle loss**

partial tissue loss of levator ani muscle, obturator internus muscle, obturator membrane, (ischio)coccygeus muscle, piriformis muscle

with

- **bare** pubis/ischium bones

**step-by-step reconstruction**

in line with the functional pelvis anatomy

this is divided into different parts

- **i** anesthesia, position, examination etc
- **ii** dye test

**specific attention post IIb (delivery) incontinence**

- **iii** incision and dissection

physiologic inverted U incision at 1 cm from euo thru bilateral deep sulci (up to superior pubis bone/ilium bone)

and
dissection of what is left of avw from what is left of pcmuf/epd

- **iv** identifying responsible defects/connection to pelvis wall

(up to extensive) anterior pcmuf/epd loss

and
total loose connection onto anterobilateral pelvis wall (atf and pubis bone body)
still some thing can be done to reinforce the continence mechanism by meticulous repair of what is left of the endopelvic diaphragm.

still do not expect too much from the incontinence operation considering the enormous tissue loss in these fistulas possible epd/pcmuf defects besides the ragged median defect aa distal or (sub)total pcmuf/epd loss bb major (sub)total loss atf/atlam over 7-7.5 cm with cc complete loose anterobilateral connection to pelvis wall dd bilateral open paravesical space possible pelvis muscle loss partial tissue loss of levator ani muscle, obturator internus muscle, obturator membrane, (ischio)coccygeus muscle, piriformis muscle with bare pubis/ischium bones step-by-step reconstruction in line with the functional pelvis anatomy this is divided into different parts i an anesthetia, position, examination etc ii a dye test specific attention post IIBb (delivery) incontinence iii incision and dissection physiologic inverted U incision at 1 cm from euo through bilateral deep sulci (up to superior pubis bone/ilium bone) and dissection of what is left of avw from what is left of pcmuf/epd iv identifying responsible defects/connection to pelvis wall (up to extensive) anterior pcmuf/epd loss and total loose connection onto anterobilateral pelvis wall (atf and pubis bone body) nb now the bilateral open paravesical space has been closed by the semicircular “anatomic” reduction/repositioning/refixation of the endopelic diaphragm onto the anterobilateral pelvis wall with normalization/repositioning of euo into its anatomic position viii check of result + fixation of indwelling catheter ix adaptation of avw x urethra tissue quality xi documentation
type kees IIBb fistulas
post IIIB incontinence

anterior trauma atf/atlam loss

anterior trauma atf/atlam loss

anterior trauma atf/atlam loss

anterior trauma atf/atlam loss
post IIb incontinence

fixation epd onto paraurethra pubis bone

fixation epd onto paraurethra pubis bone
post IIb incontinence

fixation epd onto paraurethra pubis bone

fixation epd onto paraurethra pubis bone

fixation epd onto “atf”/ischium bone

© kees
post IIb incontinence

fixation epd onto "atf"/ischium bone

fixation epd onto "atf"/pubis bone

fixation epd onto "atf"/pubis bone
post I incontinence
+ post I postdelivery incontinence

type kees I fistulas
in type I fistulas there is only tissue loss of the bladder, pubocervical musculofascia (as part of endopelvic diaphragm = epd) and anterior vagina wall (= avw) and/or cervix and/or uterus with an intact continence/closing mechanism there may be major tissue loss with also trauma to the sacrospinous ligament, (ischio)coccygeus muscles and pririformis muscles however, the urethra, urethrovessical junction and anterior part of the pubocervical musculofascia together with its connection to the paraurethra pubis bones and the arcus tendineus fasciae are still intact

post type kees I incontinence
if post type I and post type I delivery incontinence develops, probably there was already (slight) stress incontinence before due to the same defect within the endopelvic diaphragm as in genuine intrinsic incontinence or it is due to traction by fixed cervix or fibrosis into direction of the sacrum if the cause is a median longitudinal defect within the endopelvic fascia, the outcome of surgery may be good; see special chapter on genuine intrinsic incontinence however, if the cause is severe fibrosis or traction by retracted/moving cervix the outcome of incontinence surgery may be poor

possible epd/pcmuf defects

though post I repair and post I postdelivery incontinence are fairly uncommon, if it happens there are 2 possibilities which need correction

aa longitudinal endopelvic diaphragm defect with(out) cervix dis-anchoring the same defect as in genuine intrinsic incontinence with(out) dis-anchoring of cervix from within this diaphragm

bb traction onto endopelvic diaphragm by retracted/moving cervix after cs static traction by fixed cervix and/or paradoxic moving of cervix/vault on cough cephalad and anteriorly due to (in)direct adhesions to anterior abdominal wall whereby also the posterior urethra wall is pulled towards the cervix/sacrum with distortion of the urethra architecture; resulting in decreased outflow resistance
step-by-step reconstruction
in line with the functional pelvis anatomy

this is divided into different parts
i  anesthesia, position, examination etc
ii  dye test

special attention post I (postdelivery) incontinence

iii  incision + dissection
large physiologic inverted U incision at 2 cm from euo inside/parallel to ruga folds and wide dissection of avw from pcmuf/epd up to cervix

iv  identifying responsible defects epd/connection to pelvis wall
there are 2 possibilities
aa  longitudinal median defect pcmuf/epd from cervix up to 1-2 cm to euo with disanchoring of cervix
bb  severe traction onto posterior urethra wall by retracted/moving cervix/vault after cs or cs_sth or cs-tah with or without severe fibrosis with wide open euo drawn inside posteriorly

v  repair of longitudinal defect with re-anchoring cervix
aa  if median longitudinal epd defect
meticulous longitudinal repair of median defect within pcmuf/epd with proximal re-anchoring suture thru cervix
bb  if traction onto posterior urethra wall by cx/vault and/or severe fibrosis bilateral fixation of epd onto bilateral paraurethra pubis bone periost by 2x polyglycolic acid sutures each side in order to neutralize the traction by epd towards the sacrum

vi  repair of any further defect within endopelvic diaphragm
vii  refixation of any loose epd connection to pelvis wall
viii  check of result + fixation of indwelling catheter
ix  adaptation of avw
x  urethra tissue quality
xi  documentation

see also under (postpartum) genuine intrinsic incontinence
(postpartum) genuine intrinsic incontinence

with(out)
hourglass urethra
with(out)
cervix prolapse c1 or c2

genuine intrinsic incontinence is very common all over the world and poses a big challenge to the reconstructive surgeon

the difference with postrepair incontinence is that in genuine incontinence there is no anatomic tissue loss but functional defects within the endopelvic diaphragm

there are many theories to explain and even more operation techniques to handle this condition

however, the author could not comprehend these theories and considers the operation techniques as tricks though they may work

so, he thinks the concept of the corpus intrapelvinum with endopelvic diaphragm is a more scientific explanation and then used for real reconstructive pelvis surgery; see basic science

genuine intrinsic urine incontinence is frequently combined with cervix prolapse c1 or c2 in combination with wide pelvis as expressed by a pubic arch of $\geq 90^\circ$

cervix prolapse c1 and c2

introduction pop q c1 and c2

when the lowest point of the cervix comes up to 2 cm proximally from hymen ring this is called cervix prolapse c1 according to the pelvis organ prolapse quantification system

this is a frequent finding in parous females

normally this does not need surgery

when the lowest point of the cervix reaches between 1 cm proximally and 1 cm distally from the hymen ring this is called cervix prolapse c2

this may need reconstructive surgery if the woman complains about it or if combined with urine incontinence; as part of the operation technic; same as for cystocele
type kkees I fistulas

vesicovaginal fistula

vesicocervicovaginal fistula

vesicouterine fistula

vagina vault fistula
post I incontinence

cervix trauma

fascia defect type I fistula
kees endopelvic diaphragm reconstruction with cervix re-anchoring under spinal anesthesia in the exaggerated lithotomy position

001 measure pubic arch in degrees
002 measure vagina length in cm
003 suture both labia minora onto inner side of the upper legs/buttocks
004 check for urine intrinsic incontinence by asking the patient to cough
005 if necessary a median episiotomy can be performed but normally this is not indicated since wide pubic arch of ≥ 90°
006 place self-retaining auvard speculum over posterior vagina wall = pvw
007 measure distance euo to bladder wall = euo/bw in cm by metal sound
008 insert foley ch 18 catheter and drain bladder half; leave some urine inside to check later for bladder trauma (bloody urine)
009 remove foley catheter
010 measure urethra length = euo/b in cm; normally it is reduced ≤ 2 cm (vesicalization) due to rotational descent of posterior urethra wall/uv-junction/bladder base
011 then calculate longitudinal bladder diameter as euo/bw minus euo/b in cm
012 physiologic large curved incision anterior vagina wall = avw at 2 cm from euo within ruga folds towards both underlying ischium spines
013 sharp dissection of avw up to cervix so pubocervical musculofascia (as part of the endopelvic diaphragm) becomes exposed
014 identify the musculofascia; do not look for a fascia but look for smooth muscle fibers
015 measure the extent of the longitudinal median defect within the endopelvic diaphragm in cm
016 reconstruct the endopelvic diaphragm by longitudinal repair of the defect by single layer of polyglycolic acid (vicryl)
017 make sure the most proximal suture picks up the cervix as well so the cervix will be re-anchored into its anatomic position as centrum tendineum intrapelvinum
018 check if the endopelvic diaphragm is well fixed onto the pubis bones and its bilateral arcus tendineus fasciae = atf
019 if not refix the pubocervical musculofascia onto its anatomic origin: paraurethra pubis bones and paraurethra atf
check if the cervix is now mobile in its anatomic position; if not see next chapters

check for incontinence by asking the patient to cough

reinsert foley catheter

check for urine flow; catheter inside bladder, at least one urine functioning and patient not in shock

if bloody urine one knows bladder has been traumatized; this will heal by longer post operative catheterization

remove catheter

measure urethra length again

normally this will be 1-2 cm more than at the beginning (re-urethralization) since the repaired intrapelvic urogenitodigestive diaphragm ensures physiologic configuration of urine continence mechanism with normalization of outflow resistance

adapt avw by everting interrupted vicryl or nylon sutures ensuring hemostasis

reinsert foley catheter and leave it for couple of days

vagina pack up to the preference of the surgeon

operation time 20-25 min

blood loss 50-100 ml

discussion

cervix prolapse c1 and c2 are frequently found and only need surgical repair on special wish or as part of the operation technic for genuine urine intrinsic incontinence

in the 398 genuine urine incontinence reconstructions so far there was an evidence-based success rate of > 95% without intra- and postoperative complications

since the functional pelvis anatomy is reconstructed by a minimum approach step by step in a logical and systematic way using the available autologous structures

re-anchoring the cervix into the intrapelvic urogenitodigestive diaphragm is an essential part of reconstructing the functional pelvis anatomy

the tricks using artificial materials may function but these have nothing whatsoever to do with reconstructive pelvis surgery

since besides using foreign-body materials the underlying defects are not corrected so actually nothing is reconstructed
and seem to be more for the financial benefit of the surgeon and medical industry than for the well-being of the woman.

there were some failures with congenital urine incontinence but these may have been due to a concomitant neurologic component.

if not successful or if recurrence after subsequent deliveries exactly the same technique was performed again with excellent results.

in identifying the intrapelvic urogenitodigestive diaphragm (components) one has to look for smooth muscle tissue and not for fascia tissue.

since there is no anatomic tissue loss but splitting of the fibers with bilateral retraction the musculofascia structures could always be identified/found by the author and were sufficient for the reconstructive surgery.

the author uses the same technique for cystocele with or without incontinence and with or without cervix prolapse c1 and c2.

the technique as described is totally different from the anterior colporrhaphy which the author considers as rather mutilating using a mutilating longitudinal incision with poor access, just suturing some kind of unidentified tissue and removing healthy valuable tissue which is against any reconstructive surgery principle.

nb in post-fistula-repair incontinence with anatomic tissue loss one has to look for the responsible defects within the endopelvic diaphragm and then repair them all one by one meticulously.
and seem to be more for the financial benefit of the surgeon and medical industry than for the well-being of the woman.

If not successful or if recurrence after subsequent deliveries exactly the same technique was performed again with excellent results.

In identifying the intrapelvic urogeneric diaphragm (components) one has to look for smooth muscle tissue and not for fascia tissue since there is no anatomic tissue loss but splitting of the fibers with bilateral retentions the musculofascia structures could always be identified/found by the author and were sufficient for the reconstructive surgery. The author uses the same technique for cystocele with or without incontinence and with or without cervix prolapse c1 and c2. The technique as described is totally different from the anterior colporrhaphy which the author considers as rather mutilating using a mutilating longitudinal incision with poor access, just suturing some kind of unidentified tissue and removing healthy valuable tissue which is against any reconstructive surgery principle.

In post-fistula-incontinence with anatomic tissue loss one has to look for the responsible defects within the endopelvic diaphragm and then repair them all one by one meticulously.

genuine intrinsic/stress incontinence cervix prolapse c1 and c2
postrepair fibrosis incontinence
+ postrepair fibrosis postdelivery incontinence

postrepair fibrosis/traction incontinence
this is a very serious condition due to severe fibrosis/mutilation with traction onto the posterior urethra wall towards sacrum
due to the obstetric trauma, due to cesarean section with paradoxic moving on cough, but mostly due to (repeat) surgery by incompetent surgeons though it happens as well in experienced hands
and may be an additional factor in all types of postrepair incontinence from type I thru type III urine fistulas
and the great majority are inoperable
if still operable an effort has to be made to neutralize the traction by anterobilateral fixation of avw_cervix_endopelvic diaphragm onto pelvis wall under high tension
with special attention on anterior epd/pcmuf fixation onto paraurethra pubis bone periost in order to neutralize the traction towards the sacrum
good prognosis in only 10-20%

possible epd/pcmuf defects
aa only traction or up to stone-hard fibrosis
bb any defect characteristic for the fistula type I to IIBb

step-by-step reconstruction
in line with the functional pelvis anatomy
this is divided into different parts
  i  anesthesia, position, examination etc
  ii dye test
  iii incision and dissection
  iv identifying responsible defects/connection to pelvis wall
specific attention postrepair fibrosis incontinence

v + vi  fixation under tension of pcmuf/epd onto pubis bone periost
  in order to neutralize the traction

freshening paraurethra pubis bone body and bilateral fixation of pcmuf/epd under
tension onto pubis bone periost by 2x polyglycolic acid sutures each side to neutralize
traction onto the posterior urethra wall

vii  re-anchoring cervix into epd if indicated
viii check of result + fixation of indwelling catheter
ix  adaptation of avw
x  urethra tissue quality
xi  documentation
postrepair fibrosis/traction

fixation epd onto paraurethra pubis bone under tension

fixation epd onto paraurethra pubis bone under tension
discussion postrepair incontinence

the general consensus about stress incontinence is that the cause is rotational descent of the bladder neck, uv-junction and proximal urethra resulting in flattening of the angle between the bladder neck and the urethra so that the intraabdominal pressure rise is misdirected;

nb all due to levator ani muscle/pelvis floor dysfunction/trauma

and all surgical techniques are based on elevation of the bladder neck, uv-junction and proximal urethra in order to restore the angle between bladder neck and urethra

however, it is only rotational backward descent of the posterior bladder neck, posterior uv-junction and posterior proximal urethra resulting in funneling of uv-junction and proximal or whole urethra with traction upon the posterior urethra wall towards the sacrum resulting in posterior shifting of the posterior against anterior urethra wall with distortion of the urethra architecture leading to a decrease in outflow resistance and impaired intrinsic closing forces;

all due to functional and/or anatomic defects within the endopelvic diaphragm

so the author thinks another concept is needed of the corpus intrapelvinum with the endopelvic diaphragm with the cervix as its center as the main support of the urine continence/closing mechanism

instead of elevating the bladder neck, uv-junction and proximal urethra as a general trick by a multitude of operation techniques (which may work)

the author is of the opinion that the real cause(s) has/have to be identified first and then meticulously repaired using general reconstructive principles in order to reconstruct the functional pelvis anatomy ensuring physiology, ie continence

these principles have to be applied already during the first or further attempt at closing the obstetric fistula in order to prevent postrepair incontinence

however, postrepair intrinsic stress urine incontinence still develops, remember also anatomic tissue loss, even in experienced hands where these principles have been followed

and then the real cause has to be identified and an attempt made to (again) reconstruct the functional pelvis anatomy; if possible

though the operation principles are evidence-based effective, these have to be customized since each fistula and every postrepair and also genuine incontinence constitutes its own unique entity

considering the enormous variety of the complex obstetric trauma it is clear that this is highly complicated and

however skilled the surgeon is and whatever his attempt(s) there remain fistulas and postrepair incontinence problems which are right from the beginning or have become during the (repeat) attempts inoperable nothing is simple
pre- and postoperative care

importance oral fluid intake
bladder drill/training
pelvis floor exercise
postoperative instructions
importance of high oral fluid intake

the patients are highly intelligent and notice that when they drink plenty they will leak plenty and when they drink little they will leak little

so after some time, but especially when they have either long-standing fistula or long-standing (postrepair) urine stress incontinence, most of them will restrict their oral fluid intake to the minimum

however, that is one of the worst things that can happen since: urine output will be minimal and the urine concentrated resulting in:

a recurrent urinary tract infections with in the end a shrunken bladder
b infectious hydroureters with hydronephros
c stone formation
d severe urine dermatitis
e offensive odor
f more social outcast
g they give up hope since it is difficult to get cured

to operate the patients in this stage is associated with problems and bad results such as high percentage of breakdown, cystitis, uv-structre etc

therefore it is of utmost importance to rehabilitate the patient to start drinking ... and abundant drinking ... already before any repair is undertaken

they have to understand that they first have to leak more before they can be cured and this requires patient compliance

ultimately the patient is responsible for her own health and not the surgeon; it is not possible to cure an uncooperative patient

so the first thing in the management of the obstetric fistula is to explain and instruct the patient to drink at least 6-8 liters per day and make her understand that if she is not drinking there will be no operation

this will also help in the patient complying to drink postoperatively since she is already used to it; and during operation it might help in identifying the ureters

it is easy to check as one only needs the patient to stand for a couple of minutes and if no leakage tell her to come back if drinking

it will select the cooperative patients from the uncooperative patients; if the patient is uncooperative, do not operate; it is asking for trouble

theoretically there is a minimal risk of developing hyponatremia when the patient is not eating so on the operation day some salt may be added to the water

you want to be dry ... you have to comply
bladder drill/training
theory and practice
resting is rusting

theory as introduction

all body tissues/organs need to work/function regularly, otherwise they will atrophy; the more they are being used/exercised the stronger they become

and the bladder is not an exception

the function of bladder physiology is filling up to a certain point with isobaric stretching of the detrusor smooth muscle fibers and then emptying by contraction of the detrusor muscle

if somebody is not drinking sufficiently this process will be countered by atrophy of the detrusor muscle fibers with loss of function

behavior

persons with urine incontinence have a tendency to drink less thinking they will leak less urine and so control their problem better

however, in the process the detrusor muscle will atrophy and can no longer maintain its function optimally

this may explain the fact that they are no longer able to empty the bladder completely on micturition and have a urine residue

so they need to be retraining the detrusor muscle in order to restore its function

physiologic behavioral bladder training

by drinking more and urinating more frequently the detrusor muscle will be trained with stronger fibers and then it will function better

also in genuine intrinsic and postrepair intrinsic stress incontinence

practice

first of all the mechanism has to be explained; as well that first they will leak urine more before improvement is noted

and that ultimately they themselves are responsible for their own health and not the surgeon who is only helping them with their obstetric trauma health problem by his (state-of-the-art) reconstructive surgery
then they are instructed to drink more than 4,000 ml/24 hr or even more and urinate every 30-45 minutes whilst making an effort to empty the bladder completely and to continue to comply with and continue this practice leaking or not since that is in their own interest
tip
certain things work very well in community settings
so bladder drill classes were organized for all patients together in need of this training under the supervision of an attendant instructing/supervising this training whereby some patients started to instruct the others as well by copying the others
results very effective
by this bladder drill/training over 50% of the patients with immediate postrepair incontinence became completely dry within 2 weeks and another 20-30% after a longer period
observation
even if this bladder drill, as combined or not with pelvis floor exercises, fails quite a number of patients with severe incontinence become fully dry after 1-1.5 year especially when they become pregnant again subsequently
most of them stay dry
whilst some others do develop a new fistula or develop postrepair postdelivery urine incontinence

nb a pregnancy may cure postrepair incontinence though it may recur after delivery
pelvis floor exercises
mechanism of action

introduction
empirically, pelvis floor (levator ani + perineum) muscle exercises may have a positive effect upon urine intrinsic stress incontinence

however, though it is recommended everywhere by everybody the author could not find an explicit explanation for this action

functional anatomy
the pubococcygeus muscles envelop the vagina three-quarterly bilateroposteriorly as a sling being in direct contact with the bilateral vagina walls and in indirect contact (via anorectum) with the posterior vagina wall

the open anterior/cephalad one quarter gap is filled up by the symphysis and endopelvic diaphragm/pubocervical musculofascia with adherent anterior vagina wall and adherent posterior bladder base, posterior uv-junction and posterior urethra whilst

all the continence mechanisms are firmly anchored into the perineum outlet diaphragm

mechanism of action
when the pubococcygeus muscles contract they squeeze the posterior and bilateral vagina walls with the effect that the anterior vagina wall (not squeezed) moves anteriorly and cephalad together with the adherent endopelvic diaphragm/pubocervical musculofascia with adherent posterior bladder neck, posterior uv-junction and posterior urethra wall

this will reinforce the intrinsic urine continence mechanism since the posterior urethra wall will rotate forward towards the anterior urethra wall and symphysis resulting in a better arrangement/architecture of the anatomic urine continence mechanism; so a positive effect but indirectly

the simultaneous contraction of the muscles with increase in the tonus of the perineum outlet diaphragm will directly have a positive effect on all the continence mechanisms since these are firmly anchored into it

and probably increase in the tonus of the endopelvic diaphragm by smooth muscle fiber contraction by reflex action

at the same time there will be an increase in the extrinsic rhabdosphincter forces by voluntary and/or reflex contractions

however, there is no guarantee of success since it will only be successful if

now by this action the total amount of forces closing the urethra will be higher than the intravesical excretion pressure
optimal way of using pelvis floor muscles
one first contracts the pelvis floor muscles before standing up or before coughing so that the configuration of the anatomic urine mechanism is optimal just before there is an increase in (intraabdominal and so in) intravesical pressure

if one does this regularly first their action will become stronger and second our able brain will create special pathways for it and it may become a reflex

small or no effect with increasing endopelvic diaphragm defects
however, if there are defects within the endopelvic diaphragm or its anterobilateral/circumferential fixation to the pelvis wall with poor or nil support of the continence mechanism the action of the levator ani muscles will have only small or no effect since there will be insufficient or no reduction of the posterior urethra wall

discussion
it is good to realize that the pelvis floor functions as one dynamic unit with an indirect action by the levator ani muscles and a direct action by the perineum outlet diaphragm upon the urine continence mechanism

whereby the action of the levator ani muscles is reinforced by the simultaneous action of the perineum outlet diaphragm and the other way round since

they are firmly connected to each other via the perineal body, the external sphincter ani complex and the levator plate

with perineal body as centrum tendineum perinei since all the relevant structures are firmly anchored into it

caution
it is only in mild forms of intrinsic stress incontinence that the pelvis floor exercises may have a beneficial effect for a short time

however, there are no randomized controlled studies available claiming a long time beneficial effect

the function of the levator ani muscles in preventing and treating intrinsic stress incontinence as main support of the urine continence mechanism are highly overvalued

and another concept of securing/stabilizing the pelvis organs and their continence mechanisms is needed as

corpus intrapelvinum with endopelvic diaphragm
The optimal way of using pelvic floor muscles is by first contracting them before standing up or before coughing, so that the configuration of the anatomic urine mechanism is optimal just before there is an increase in intraabdominal and intravesical pressure. If this is done regularly, first, their action will become stronger, and second, the brain will create special pathways for it and it may become a reflex.

However, if there are defects within the endopelvic diaphragm or its anterobilateral/circumferential fixation to the pelvis wall, there may be poor or nil support of the continence mechanism, which means that the action of the levator ani muscles will have only a small or no effect, since there will be insufficient or no reduction of the posterior urethra wall.

Discussion
It is important to realize that the pelvic floor functions as one dynamic unit with an indirect action by the levator ani muscles and a direct action by the perineum outlet diaphragm, wherein the action of the levator ani muscles is reinforced by the simultaneous action of the perineum outlet diaphragm and vice versa, since they are firmly connected to each other via the perineal body, the external sphincter ani complex, and the levator plate with perineal body as centrum tendineum perinei since all the relevant structures are firmly anchored into it.

Caution
It is only in mild forms of intrinsic stress incontinence that the pelvic floor exercises may have a beneficial effect for a short time. However, there are no randomized controlled studies available claiming a long time beneficial effect. The function of the levator ani muscles in preventing and treating intrinsic stress incontinence as main support of the urine continence mechanism are highly overvalued, and another concept of securing/stabilizing the pelvic organs and their continence mechanisms is needed as corpus intrapelvinum with endopelvic diaphragm.

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postoperative instructions and follow-up

postoperative ward

check blood pressure and pulse every 30 min for 4 hours

if history of eclampsia administer 10 mg valium im the afternoon of operation

liberal use of analgesics

if no urine on operation day start iv fluids with lasix iv and call the doctor

encourage **oral fluid intake, at least 6-8 liters per day**

if not drinking, vomiting or low urine output start iv fluids

feeding as soon as patient feels like it

check catheter drainage, and if blocked flush it or change it

**urine should be at least 4,000-6,000 ml per 24 hours and completely clear**

full mobilization the morning following surgery

no antibiotics unless specifically ordered

fersolate and folic acid 1 tablet ods each

if indicated iron dextran im

remove pack after 1 day, carefully otherwise the patient will start bleeding

if fever develops give antimalarials as well and do the necessary

episiotomy sutures to be removed after 7-10 days

catheter to be removed after 2-4 weeks in the operation theatre

after removal of the catheter the patient is discharged from the postoperative ward back to the hostel; she has to be instructed to continue drinking and to pass urine every 10-15 minutes

nonabsorbable intravaginal sutures to be removed 1 week after catheter removal; that is also the first postoperative intravaginal examination

**additionally for rvf**

**no** solid food for 10 days

laxatives like liquid paraffin for 10 days

**no** sitzbaths unless wound becomes septic

after defecation cleanse anus/perineum with water/soap and wipe dry and keep dry
postoperative follow-up

nonabsorbable intravaginal sutures to be removed 1 week after catheter removal

then 2 weeks later check-up

then 1 month later check-up

then 2 months later check-up

then 2-3 months later last check-up; patient can resume sexual activities

at each check-up please ask for the following and do not forget **health education**:

leaking yes/no for rvf passing stools/flatus per vaginam

incontinence yes/no for rvf stool/flatus continence

normal miction yes/no for rvf normal defecation

then examine and check for the following:

healed yes/no for rvf perineal body/sphincter ani

elevation good/moderate/poor

(stress) incontinence yes/no

if reporting with pregnancy following (successful) repair give health education and set date for **elective cesarean section**

whatever you do write it down for documentation
prevention of postrepair incontinence at first or further repair

obstetric fistula only by establishing a network of functioning obstetric units some 150,000 in Africa only
prevention
postrepair incontinence

prevention of postrepair incontinence
  at first or further repair

prevention
obstetric fistula

only by establishing a network of
  functioning obstetric units
    some 150,000 in africa only
prevention postrepair incontinence
already at first attempt

introduction

the best chance of continence after vvf-repair is when the (obstetric) fistula is closed within the whole process of reconstruction of the functional anatomy in the complex obstetric trauma since the obstetric fistula is part of the complex obstetric trauma already during the first repair elements have to be built in to reconstruct the functional pelvis anatomy with in the process closure of the fistula to ensure continence by repairing the longitudinal, transverse, quartercircular or semicircular defects within the endopelvic diaphragm and if necessary refixation of the diaphragm to the pubis bone and atf and if necessary by re-anchoring the cervix with closure of the fistula within the reconstructive surgery process so the complicated functional pelvis anatomy in the female has to be studied and mastered fully since the intact endopelvic diaphragm is the decisive factor securing the bladder and cervix in their anatomic position and so supporting the continence mechanisms it is important already during the first attempt to check systematically for (different) defects within the endopelvic diaphragm and when found to repair these meticulously this has to be checked at operation ending by the fact that the urethra and euo will be of normal width in the anatomic position

the following will describe these defects in the different fistula types from type kees I to type kees IIBb

type kees I fistulas
though the anatomic continence mechanism is not involved in the type I fistulas there may be a longitudinal diaphragm defect and the anchoring of the cervix may be loose this has to be checked during the repair and then repaired meticulously

type kees IIa fistulas
there is always a kind of transverse anatomic defect (tissue loss) within the endopelvic diaphragm; if large there is indirectly no connection to the pubis bones and this has to be repaired as well to make sure the endopelvic diaphragm with its circumferential connection to the pelvis wall is reconstructed
type kees IIAb fistulas
there is always a quarter- or semicircular defect with tissue loss within the endopelvic diaphragm with some kind of up to extensive arcus tendineus fasciae tissue loss

and all these have to be repaired meticulously to ensure that the endopelvic diaphragm with its circumferential connection to the pelvis wall is reconstructed together with the circumferential end-to-end vesicourethrostomy

type kees IIBa fistulas
there is a median distal or larger defect with tissue loss within the endopelvic diaphragm with indirect loose connection to the anterior/anterolateral pubis bons/atf due to posterolateral retraction

and after longitudinal inverted T urethra reconstruction the endopelvic fascia has to be refixed onto the paraurethra pubis bones and paraurethra atf over the reconstructed urethra

type kees IIBa fistulas
normally there is extensive tissue loss of the urethra and endopelvic diaphragm and need a two-stage repair

already at first stage the endopelvic diaphragm has to be refixed to the para-“euo” pubis bones and atf from ischium spine, if possible, to para”urethra”-“euo atf together with circumferential fixation of the bladder neck into “euo” converting the type IIBb into type II Ba and

then if necessary at second stage the remaining defects have to be repaired

evidence-based (in)continence results
the (in)continence results are progressively worse from type I thru type IIBb since and the anatomic continence mechanism and the support by the endopelvic diaphragm are progressively more traumatized with more tissue loss

if the result is persistent urine intrinsic-stress incontinence, even after bladder drill and pelvis floor muscle exercises, an incontinence operation is indicated as outlined
prevention obstetric fistula

if you wanna work……work……don’t talk

when we listen to the political rhetoric of the major aid organizations the obstetric fistula will be eradicated by the year 2030 employing legislation for age at marriage, human rights, gender issues, empowering women, culture change, empty declarations etc etc, the one even more noble than the other

however, they keep on talking, are not listening to the professionals and are so busy by praising themselves into heaven they forget to implement the real and only strategy

setting up a network of functioning obstetric units throughout africa and asia only for africa some 150,000 where at any time a cesarean section can be performed one by one on a 100,000 mile road

once the service is reliable the public will rush to use this service since healthy mother and healthy child

forget about the politics and start the pragmatics and let the money roll

first the hardware
set up small operating theater for cesarean section only as equipped with high-quality operating table high-quality operating lights high-quality instruments small standby generator for theater only reliable fuel supply reliable water supply emergency drugs high-quality consumables set up blood bank costs per unit some U$ 50,000-100,000

second the software
at the same time prepare the software for a 24-hour non-stop service train/select/maintain professionals: doctors, nurses and other personnel needed per unit 4-5 surgeons, 4-5 anesthetists, 10-15 nurses and other staff costs per unit ????

infrastructure
build more roads

responsiblity
politicians, aid organizations, professionals, urban and religious leaders, the general public and last but not least the women themselves

so when are we wakening up and stop talking and start working and doing what we have to do on a long and curvy road so that by the year 2100 we may have an impact
incurable incontinence

protocol incurable fistulas

incurable postrepair incontinence

postdiversion urine/stool incontinence
incurable fistula patients  kees protocol
characteristics and how to behave

introduction
how capable the surgeon may be there are always patients in whom the trauma is right
from the beginning or has become during surgical intervention(s) incurable

what is incurable
incurable means that
either
the surgeon is no longer able to operate anymore
or
the patient will not benefit anymore from an operation
or
the operation would endanger her health and/or her life

who will encounter it
for the inexperienced surgeon it comes at an early stage and for the experienced
surgeon it comes at a later stage
however, any surgeon, whatever his experience and skills, will encounter patients who
are beyond repair

characteristics
absolute
valid for any surgeon whatever his experience
no tissue left to reconstruct like (sub)total bladder/urethra loss
everything fibrotic/fixed so the tissue cannot be mobilized
relative
depending upon the surgeon’s experience
severe vagina stenosis
severe mutilation  either obstetric or iatrogenic or combined
extensive obstetric trauma like in type IIb
repair “possible” with predictable resulting incontinence if closed
total post IIAb/IIb intrinsic incontinence
procedure which would take more than 2-3 hours
severe obesity with fistula deep inside
old age with long-lasting urine leaking

aetiology
minority due to extensive obstetric trauma and/or procedures by experienced fistula
surgeons
majority due to mutilating procedures by (in)experienced (fistula) surgeons who do not
understand the obstetric trauma and/or pelvis anatomy due to lack of training and/or
self-arrogance
perils of obstetric fistula surgery
the surgical management of the obstetric fistula surgery will bring down any surgeon from whatever background, training, race or country down to the earth with both feet planted solidly on the ground since anybody (even the blind) can see (or smell) the misfortunes however much the surgeon is boasting about his performance and then even in highly competent hands: operation successful, patient leaking

main problem
not the fistula itself; since closure rate is up to 95-96% but how to ensure/secure social continence in order for her to live a “normal” life whatever that may be

how to behave in order to prevent iatrogenic mutilation
any surgeon whatever his experience should ask him/herself
a will the patient benefit from my surgery
b can i handle it safely
c how do i obtain quality in my surgery
d can i handle it myself or do i refer to a more competent surgeon
e is it not shameful to do something i know deep inside i cannot handle

who/how to determine incurable
the most experienced fistula surgeon in a certain area is the one who should determine “incurable”
however, since the obstetric and/or surgical trauma is so complex this can only be done by: an examination under (spinal) anesthesia and then the surgeon should proceed if possible or stop if really incurable and document what is found for himself and others

how to behave if incurable
the situation has to be explained to the patients (and her relatives) in so far as she is able to understand it she has to be instructed in personal hygiene and drinking she has to be taught skills to look after herself; she has to come once a year for check-up and to see if new techniques have become available do not lock her up in a fistularium not even in a golden prison since ultimately the patient herself is responsible for her own life

is incurable incurable forever
incurable does not as a rule mean incurable for ever since the responsible surgeon may gain the additional expertise new insights with new techniques may become available with time another surgeon may have the necessary expertise
personal experience
after over 25,000 repairs the author still faces incurable patients in 0.5-1% with an upward trend since more and more patients are referred to him and more and more inexperienced “surgeons” operate
in the very beginning the patients were told to come back after 1 year and with more experience most of them were operated successfully
now we are in the process of reviewing them all one by one to execute the final last resort assessment of possibilities
with promising results since we developed new techniques for post-repair incontinence

upward trend of incurability
since obstetric fistula became all of a sudden sexy around the year 2000 more doctors and more organizations became interested
however, they are interested more in quantity than in quality for short-term show-off in order to raise funds; how can one train 120 doctors surgically in only 80 patients
this resulted in an increase of ill-trained “surgeons” with mutilating techniques thanks to the arrogant interference by people who are only interested in political rhetoric

conclusion
since fistula surgery is highly complicated, the majority are the result of poor surgery by incompetent (and arrogant) surgeons; one “repair” can spoil the patient’s life forever
any surgeon should know his limits and restricts him/herself to things (s)he is sure (s) he can handle

too many doctors are poorly trained and as such do not have the slightest clue about the obstetric fistula and pelvis anatomy; they are only trying to copy a trick
we should all aim at quality and not at quantity

half knowledge is extremely dangerous
one repair by an incompetent surgeon may change the prospect of the fistula from operable into inoperable with devastating life-long consequences for the patient
irrepairable incontinence surgery + ba hanya
advanced level

though progress has been made in preventing and treating postrepair incontinence irreparable incontinence still continues to be the major challenge which has not yet been solved

mainz II pouch
is a complicated procedure, needs intensive postoperative care, carries a high morbidity and mortality rate in the developing world and is scientifically not sound since pouch not a reservoir since water/urine goes immediately to lowest point, ie the rectum, as any plumber will tell
pouch not low pressure since then stool from the proximal and distal loop would accumulate in it and the pouch would rupture within a couple of days
extramural ureter implantation a myth since at one point the ureters must pass thru the rectum wall
using the digestive tract for urine storage is nonphysiologic and making the woman chronically “diarrheic” by mixing the distal stool with urine and lastly many patients are very unhappy with it especially the nightly soiling and “shitting like a chicken”

ureterosigmoidostomy
far less complicated and far less traumatizing; however with same outcome

mitrofanoff principle
this seems to be a better option since not so complicated and urine storage stays inside the bladder as physiologic reservoir
however, late stenosis of appendix skin opening may develop and it needs good patient compliance since 3-5 times daily self-catheterization to empty the bladder completely, otherwise stone formation may be the result

surgery for severe vagina stenosis (ba hanya)
kees neovagina

needs serious patient compliance since newly created vagina has to be kept open

sigmoid neovagina
this is an established procedure; however, it is complicated with grave complications, needs general anesthesia and requires ample experience in colorectal and reconstructive pelvis surgery

creation of kees neovagina using the vagina route
the author has developed a safe technic based on the functional pelvis anatomy with good results however, it requires exact theoretic and practical anatomic knowledge and advanced level surgical skills
post diversion urine/stool incontinence

introduction
though urinary diversion into digestive tract is considered to be the final solution there are quite a number of patients ending up with urine/diarrheic stool incontinence thru the anus

especially nightly soiling which is very embarrassing to them and several patients came begging to undo this into the original situation

cause
the rectum has become the new urine reservoir since it is the lowest point of the digestive tract; and water/urine will flow immediately to the lowest point
due to fluid contents of the rectum, a situation comparable to diarrhea, also reactive rectum muscle contractions trying to expel this watery content

insufficient stool continence mechanism with(out) poor support by (insufficient) perineal body

even if the woman can hold it during daytime when fully awake, at night during sleep the sphincter ani complex will relax resulting in urine/stools soiling

surgery
the aim is to strengthen the stool continence mechanism by

quarter- or semicircular incision at anterior anus

sharp dissection over sphincter ani muscle

if possible rhaphy of preanorectum fascia/muscularis over 2-4 cm so it becomes narrow with stronger closing force

rhaphy of sphincter ani by 2-3 sutures so it becomes narrow with stronger closing force

longitudinal reinforcement of perineal body by 2-3 sutures for better support

if necessary longitudinal adaptation of perineum skin
general discussion

postrepair incontinence
cause
management
prevention
general discussion
postrepair urine incontinence, cause, management etc

postrepair incontinence
postrepair incontinence and postrepair postdelivery incontinence are common and pose a challenge to the obstetric trauma surgeons, inexperienced and experienced
this is the first scientific approach to this major problem

classification
a scientific classification is presented as based on the classification of the original fistula with is different anatomic tissue loss characteristics of the endopelvic diaphragm and its anterobilateral fixation to the pelvis wall

cause
the real causative factors are analyzed with special attention to the functional and anatomic defects within the endopelvic diaphragm and its semicircular anterobilateral fixation to the pelvis wall, arcus tendineus fasciae and pubis bone bodies

another concept of the corpus intrapelvinum with endopelvic diaphragm is presented in order to provide a better understanding

the role of the levator muscles in urine (in)continence is highly overrated since there is no anatomic connection whatsoever between these muscles and the urine continence mechanism, bladder neck, uv-junction, urethra

management
conservative management by bladder drill with(out) pelvis floor muscle exercises, though there exists no validated evidence for the latter

surgical management by a causal approach by first identifying and then meticulous reconstruction of the endopelvic diaphragm and its connection to the pelvis wall by the repair of any defect within the endopelvic diaphragm and its connection/suspension onto the pelvis wall if possible

all in order to restore the support of the continence mechanism by the endopelvic diaphragm with (re)urethralization of the bladder neck/proximal urethra resulting in “normalization” of the urethra architecture so the intrinsic forces will be reinforced with an increase in outflow resistance
all the other approaches are just tricks to elevate the bladder neck to improve the angle between the bladder neck and the urethra, like slings, tvt, tot; though they may work but over 20% result in major complications

distal urethra lengthening beyond the anatomic euo position will not work since it is proximal urethra lengthening with full support which is needed

prevention

reconstruction of the functional pelvis anatomy at first or further attempt by addressing the complex obstetric trauma as a whole with in the process closure of the fistula

however, this requires another surgical philosophy with a change from quantity to quality surgery

where simple things are only for simple surgeon dummies who simply do not follow-up their own simple handwork and will stay simple for the rest of their simple life
basic science

see textbook functional female pelvis anatomy

urine incontinence mechanism

endopelvic diaphragm + defects

another concept
mechanism of urine stress incontinence

genuine and post fistula repair

introduction

if one wants to perform reconstructive surgery one must know the anatomy and the mechanism of action of continence and of incontinence

here a short outline is given about the mechanism of urine intrinsic stress incontinence either in genuine incontinence or post fistula repair stress incontinence

as based upon the systematic examination/assessment/documentation/analysis of various defects and tissue loss of the continence mechanism from one cell to total loss as a “natural experiment” within the complex obstetric trauma

stress incontinence is always an expression of defective intrinsic forces, in genuine incontinence without tissue loss and in post fistula repair with anatomic tissue loss

physics of incontinence

once the intravesical excretory pressure becomes higher than the closing forces of the urethra there will be outflow of urine thru urethra either voluntarily as normal micturition or involuntarily and then it is called incontinence

mechanism of urine intrinsic stress incontinence

there are 3 mechanisms of action, either isolated or combined

   aa downward force with dislocation of posterior urethra wall, uv-junction, bladder neck

   bb traction onto posterior urethra wall towards cervix/sacrum

   cc dislocation of distal posterior urethra wall/posterior euo out of its anchoring

aa vesicalization of proximal, mid- or whole urethra

the “immobile” anterior urethra wall is adherent/sticking to the posterior symphysis by loose connective tissue and a thin fluid film which allows the anterior urethra wall to slide/shift against the symphysis friction free, though little; however it cannot rotate backwards away from the symphysis also since it is pressed against the symphysis by hydrostatic pressure

the mobile posterior urethra wall is firmly adherent to the pubocervical musculofascia as part of the endopelvic diaphragm
if defects develop within this diaphragm the posterior urethra wall will rotate backwards away from the symphysis causing partial (or total) funnelling of the proximal (eventually whole) urethra since the anterior urethra wall stays sticking against the symphysis; this process is called vesicalization since functionally the funneled (part of the) urethra becomes part of the bladder (vesica)

this first force is downward due to herniation of the posterior bladder/posterior bladder neck/posterior uv-junction/posterior proximal urethra thru the median defect in the endopelvic diaphragm as seen in cystocele

or due to a loose endopelvic diaphragm since its connection to the pubis bone has been lost either directly as in circumferential fistulas or indirectly by a transverse defect

bb shifting of posterior urethra wall with distortion of architecture

besides backward rotation there is also backward shifting of the posterior urethra wall against the anterior urethra wall into the direction of the sacrum; since the anterior external urethra opening is fixed and immobile

resulting in deformation of the euo whereby the posterior euo is being drawn inside with the anterior euo still in its anatomic position

this second force is posterior into the direction of the cervix/sacrum due to pull by the herniated and/or sagging down posterior bladder wall

or due to traction by the endopelvic diaphragm when its anterobilateral fixation to pubis bones/atf has become defective

or due to scar tissue/fibrosis of anterior vagina wall, eg after mutilating longitudinal incision (anterior colporrhaphy, yankan gishiri scarification)

this second force can be the main mechanism of incontinence as seen when a longitudinal median scar from the external urethra opening to cervix (see mutilating incision) keeps on contracting throughout life since it is perpendicular to the ruga folds

it can also been seen after a cesarean section whereby the cervix is fixed intraabdominally and moves paradoxically cephalad on cough with posterior traction onto endopelvic diaphragm

it is seen frequently in ureter fistulas type III due to its posterior traction effect upon the endopelvic diaphragm

once there is vesicalization a downward force will come in as well

cc dis-anchoring distal posterior urethra wall/posterior euo

the third possibility is a trauma to the anchoring of the distal urethra and its external opening into the perineum outlet diaphragm; with or without an avulsion of the distal urethra and its external opening which may result in hourglass urethra
the external urethra opening is anchored into the perineum outlet diaphragm and anteriorly firmly connected to the symphysis whilst the posterior distal urethra wall and posterior euo are dis-anchored

total avulsion is seldom/rare though it is possible

these first two mechanisms of pathophysiologic action result into a wider (proximal) urethra lumen and a more oval elliptical arrangement of the smooth muscle fibers and interfere with the intrinsic forces keeping the urethra closed since more force is needed to close the urethra; combined with less outflow resistance according to poiseuille law

once the intrinsic forces can no longer keep the urethra closed sufficiently this will lead to genuine intrinsic stress incontinence in various degrees

in total intrinsic stress incontinence there is continuous leaking of urine on lying/sitting/standing/walking due to total loss of the intrinsic forces

intraoperatively under spinal anesthesia in these patients in the exaggerated lithotomy position the level of urine within the urethra is noticed in concord with respiration, rising on expiration and lowering on inspiration

the anterior urethra, anterior uv-junction and anterior bladder neck are connected/firmly adhesive to the posterior/inferior symphysis by loose connective tissue and thin fluid film and

the anterior bladder is connected/firmly adhesive to the posterior symphysis and the anterior abdominal wall by loose connective tissue and thin fluid film

in the upright position the anterior bladder neck, anterior uv-junction and anterior urethra wall are resting upon and pressed against the posterior symphysis

as such the anterior urethra, anterior uv-junction and anterior bladder can shift friction free against the posterior symphysis; but they cannot rotate backwards away from it due to the strong negative pressure exerted within the thin fluid film on pull/traction

there are two forces at work which exert traction upon the mobile posterior uv-junction and posterior urethra wall whereby the architecture of the urethra wall will be distorted

first the uv-junction and proximal urethra are pulled and pushed open and the urethra becomes functionally part of the bladder (vesicalization)

as long as the remaining intrinsic continence mechanism is strong enough the woman is still continent but once the intrinsic continence mechanism cannot cope any more with increased intravesical pressure there is urine loss

though this is called genuine stress incontinence actually it is intrinsic incontinence

later there will be opening up of the whole urethra (total vesicalization);
the mobile posterior urethra wall is pulled/pushed away from the immobile anterior urethra wall opposite to the direction of coaptation

besides this the posterior urethra wall is pulled towards the cervix/sacrum as well with posterior deformation of the external urethra opening so that the smooth muscle fibers become more oblique and continent closure is no longer possible and the woman looses urine more or less continuously whilst lying/sitting/standing/walking, with or without spontaneous miction

defects within the endopelvic diaphragm

there are defects without tissue loss as in genuine incontinence and defects with anatomic tissue loss as in post fistula repair incontinence

- in genuine incontinence without tissue loss
  normally one will find a median longitudinal defect whereby the posterior urethra wall is no longer supported
  however, transverse defects with indirect loose connection to the pubis bone and arcus tendineus fasciae = atf may also be found
  and direct loose connection to the pubis bone
  either isolated or combined; as well in combination with urogenital prolapse

- in post fistula repair incontinence with anatomic tissue loss
  the variety is great as is the variety in anatomic tissue loss
  median longitudinal defects with loss of support of the posterior urethra wall
  transverse defects with indirect loose connection to pubis bones/pelvis wall
  quartercircular defects with (in)direct loose connection to pubis bones/pelvis wall and loss of atf
  semicircular defects with (in)direct loose connection to pubis bones/pelvis wall and loss of atf and atlam and cephalad part of levator ani muscles etc
  (sub)total loss with major pelvis soft tissue loss
  the more extensive the anatomic tissue loss the more chance of incontinence

all these defects may be combined with anchoring defects into the perineum outlet diaphragm

defects within the anchoring into the perineum outlet diaphragm

since the anterior external urethra is firmly fixed and as such the anterior urethra wall also secured defects will lead to shifting of the posterior urethra wall and external opening towards the sacrum with wide opening of the external urethra opening and hourglass or sandglass deformity of the urethra

this anchoring defect is normally combined with a median longitudinal defect within the endopelvic diaphragm with vesicalization of proximal (+ mid) urethra

this is a rather frequent finding especially in fresh postpartum genuine incontinence

however, total avulsion of the distal urethra and external opening out of its anchoring is rare though it may occur

the author encountered this only 4 times, once due to direct trauma, once due to infection and twice due to obstructed labor
discussion and practical consequences

the obstetric fistula surgeon is in a unique position to study the female urine continence mechanism by direct observation of an endless variety of the natural experiment of the complex obstetric trauma in all its forms

the term intrinsic stress incontinence is preferred above stress incontinence since it is the intrinsic continence mechanism which is defective and has to be corrected

the art of reconstructive surgery is to first assess the trauma and then to reconstruct only the functional anatomy so that physiology will be restored under physiologic stress

since any patient with urine incontinence is unique, once the general principles have been outlined the operation technique has to be customized to correct the specific individual lesions; a standard trick may work but it is insight that counts

for intrinsic-stress incontinence a physiologic reconstructive operation technique has been developed which only corrects the defects in the endopelvic diaphragm with tightening if necessary

these principles may be of value to the industrialized world as well since most operation techniques are tricks and nonphysiologic

for all fistulas type kees I; type kees IIa, type kees IIb, type kees IIb and type kees IIb operation principles have been developed to correct the respective defects in the endopelvic diaphragm and its fixation already during the repair to prevent postrepair incontinence
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vesicalization proximal urethra

total vesicalization

downward/backward force combined

total vesicalization

anchoring trauma perineum outlet diaphragm

hourglass urethra
endopelvic diaphragm

in the female

with cervix as its central point

introduction

the intrapelvic organ and organ support situation in the female differs radically from the situation in the male by the interposition of the large female genital tract in between the distal urinary tract anteriorly and the distal digestive tract posteriorly

all embedded into the corpus intrapelvinum of the tela urogenitalis, together with their vascular and nervous supply

though the situation of the superior layer of pelvis floor is more or less the same since the levator ani muscles are not affected; except for a wider pelvis

the perineum outlet diaphragm is severely weakened by the large vagina opening; so instead of two now a third and large opening has been pierced thru punched out

so the pelvis floor in the female is prone to dysfunctioning

there is increased hydrostatic intraabdominal pressure due to the weight of the female genital organs; especially during pregnancy

also the support of the anatomic female urine continence mechanism changed since in the male it is well supported by the prostate

as compensation in order to support the female bladder and urethra and the uterus and cervix and to withstand the intraabdominal pressure the corpus intrapelvinum formed a functional dynamic structure as the author would like to call the

endopelvic diaphragm

from the pubis bone bodies anteriorly to the sacrum posteriorly and circumferentially connected to the pelvis wall like the skin of a drum or trampoline with the cervix as its center; and fusing anteriorly with the perineum outlet diaphragm under an angle of 35-40°

in between the distal urinary tract, the proximal genital tract, the intraperitoneal contents and the distal digestive tract and the vagina

with a small opening anteriorly for the urethra and a larger one posteriorly for the rectum

since the cervix is firmly anchored into the central pierced thru punched out opening it becomes the centrum tendineum intrapelvinum as well; since all the musculofascia structures are firmly anchored onto it
it consists of a mixture of connective tissue for strength, elastin for passive elasticity and plasticity and smooth muscle fibers for active dynamic non-fatigue tonus and relaxation via baro- and stretch receptors as modulated by the autonomic nervous system

it is the first line of counteracting the hydrostatic intraabdominal pressure and contributes to compression pressure by increase or decrease of its tonus; especially since its main component is smooth muscle fibers

whilst the rest pressure is dealt with by the pelvis floor structures, especially by the perineum outlet diaphragm

it supports the posterior urethra, posterior uv-junction and posterior bladder neck in their anatomic position and as such contributes to the anatomic urine continence mechanism

it prevents the posterior urethra, posterior bladder, cervix, intraperitoneal contents and anterior rectum from herniating into the vagina

it is divided into specialized parts as the pubovesical/posterior pubourethral ligaments, pubocervical musculofascia, arcus tendineus fasciae, cardinal and broad ligaments, rectovaginal fascia and sacrouterine ligaments with the cervix as centrum tendineum intrapelvinum since all its musculofascia/ligament structures are firmly connected to it

**pubovesical/posterior pubourethral ligaments (= muscles)**

anchoring the most anterior part of the pubocervical musculofascia as part of the endopelvic diaphragm onto the pubis bone bodies and

securing the posterior proximal urethra, uv-junction and bladder neck in their anatomic position and so supporting the female urine continence mechanism

once they become defective intrinsic stress incontinence may develop

**pubocervical musculofascia**

like a triangle from the pubis bone bodies and bilateral aff to the cervix as the anterior part of the endopelvic diaphragm as part of the corpus intrapelvinum

this thick musculofascia is well developed and seems to consist of longitudinal smooth muscle/collagen fibers (from anterior towards posterior) and underneath the mid/distal urethra also transverse smooth muscle/collagen fibers (in between the median inferior surfaces of the pubis bones) interwoven by collagen and elastin

the longitudinal arrangement seems likely since longitudinal median defects are found intraoperatively at genuine incontinence, cystocele and cervix prolapse surgery

the anterior transverse arrangement seems likely since the median longitudinal defects stop at 1.5-2 cm to the external urethra opening where the endopelvic diaphragm fuses with the perineum outlet diaphragm

the intact pubocervical musculofascia secures and stabilizes the (posterior) bladder base/neck, uv-junction and urethra in their anatomic position and as such supports the female urine continence mechanism; it also stabilizes the cervix anteriorly and bilaterally

the intact pubocervical musculofascia prevents the pre/subperitoneal contents bladder base/uv-junction/urethra and the cervix from herniating into the vagina
the **axis** of the pubocervical musculofascia as to the horizontal/ground is 25-30° from symphysis to ischium spine in the upright position

the posterior wall of the urethra, uv-junction and the bladder trigonum are not expanding during the asymmetric filling of the bladder; therefore these structures are firmly fixed to the pubocervical musculofascia/endopelvic diaphragm whilst

the anterior vagina wall is rapidly expanding and deflating with shearing during sexual intercourse and even more during childbirth and as such is loosely connected/fixed to the pubocervical musculofascia/endopelvic diaphragm

**arcus tendineus fasciae = atf**

as bilateral fixation/insertion of the endopelvic diaphragm/pubocervical musculofascia whilst

the arcus tendineus fasciae is further connected to the lateral pelvis wall (arcus tendineus of levator ani muscle and oburator internus muscle fascia) via a narrow triangular fascia sheath

**cervix**

the cervix is considered to be the centrum tendineum intrapelvinum since all musculo fascia structures of the endopelvic diaphragm are firmly anchored onto it and the cervix itself is firmly anchored into the central pierced thru punched out opening within the endopelvic diaphragm

**cardinal ligaments and broad ligaments**

since their smooth muscle fibers radiate into the cervix they support the endopelvic diaphragm restricting its downward movement

**sacrouterine ligaments = rectouterinus muscles**

as posterior fixation of the endopelvic diaphragm onto the sacrum since they fix/connect the cervix posteriorly onto the rectum and sacrum restricting its anterior movement

with lateral fixation to the pelvis wall (coccygeus muscles, sacrospinous ligaments and priformis muscles) via fascia sheaths

they contract during childbirth keeping the cervix in its position by preventing upward movement

**(part of the) rectovaginal fascia**

In between the vagina and rectum and anchored onto the posterior cervix in between the sacrouterine ligaments as part of the endopelvic diaphragm

**weakest point in endopelvic diaphragm/pubocervical musculofascia**

considering the anterior cone-like triangular shape with the narrowest at the pubis bones and the broadest in between the ischium spines the weakest point is in the median at the anterior cervix

and the **broader the pelvis** (with broad span) the **more prone for median defects** and as such for stress incontinence, urethrocele, cystocele and cervix prolapse

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innervation

by intrinsic myogenic impulses from baroreceptors for tonic action for a long-standing non-fatigue tonus to counteract the intraabdominal hydrostatic pressure and from stretch receptors for an immediate phasic action upon stretch on sudden intraabdominal pressure rise as superimposed upon the already existing tonic action as modulated

by extrinsic impulses from the autonomic nervous system via complex mechanism of reflex action and from hormones and from other neurotransmitters

it is very well possible that there are also impulses from the enteric nervous system

mechanism of physiologic action

the endopelvic diaphragm is a single-unit smooth muscle structure with tonic action for a non-fatigue tonus to counteract the hydrostatic pressure and immediate reactive phasic action by contraction upon stretch as superimposed upon the tonic action to counteract sudden intraabdominal pressure rise, like cough, standing up etc

this in combination with the synchronized multi-unit archaic matrix of the corpus intra pelvinum with its other single-unit smooth muscle specialized structures and as such stabilizing/securing the pelvis organs in their variable anatomic position and supporting the continence mechanisms especially preventing the posterior bladder neck, posterior uiv-junction and posterior urethra wall from backward rotation with funneling of the urethra

this explains the fact that even under spinal anesthesia and with a filled bladder a sudden fist push onto the suprapubic lower abdominal wall combined with coughing does not result in urine loss thru the euo (as stress incontinence) when the endopelvic diaphragm is intact

this test is a standard procedure to check continence during all our reconstructive pelvis surgery

reaction to biomechanic stress and hormones

during pregnancy there is continuously increasing intraabdominal hydrostatic pressure combined with hormonal flooding

since smooth muscle cells are also capable to multiply if the need arises the endopelvic diaphragm will then react by hypertrophy and hyperplasia according to the increasing intraabdominal hydrostatic pressure and hormones
during the puerperium the endopelvic diaphragm will involute as well according to the decreasing intraabdominal hydrostatic pressure until an equilibrium has been achieved

**nb** the decrease of estrogens in the second half of the cycle may explain the fact that the symptoms of stress incontinence may worsen during the second half of the cycle

**mechanism of pathophysiologic action**

the downward intraabdominal pressure upon the endopelvic diaphragm may lead to defects within this diaphragm

the downward pressure increases during the course of pregnancy with highest pressure at the median where the cervix is anchored into the endopelvic diaphragm

the broadest part of the endopelvic diaphragm is in between the ischium spines where it stabilizes and secures the cervix and

this is exactly where splitting/division of the longitudinal smooth muscle/collagen fibers at the median starts and then continues from proximally to distally whilst

the endopelvic diaphragm fibers retract bilaterally since medially disrupted

normally the most distal 1-2 cm stay intact since the short span is able to withstand the pressure and the smooth muscle/collagen fibers are also transverse (and longitudinal)

it is good to remember that during childbirth itself the pressure changes from downward caudad to upward cephalad and that semicircular compression and shearing occur at where the endopelvic diaphragm is attached to pubis bone and atf

in prolonged obstructed labor pressure necrosis may develop and lead to anatomic tissue loss defects at any location within the endopelvic diaphragm

then there may be direct trauma (penetration, surgery) and trauma due to infection

**defects within the endopelvic diaphragm**

there are two types of defects viz defects **without** anatomic tissue loss like those due to intraabdominal pressure or shearing and defects **with** anatomic tissue loss varying from minimal to (sub)total loss like those due to pressure necrosis in prolonged obstructed labor or due to infection or due to surgery

**aa** defects without anatomic tissue loss

since it is the first line of withstanding intraabdominal hydrostatic pressure especially during pregnancy and also withstanding shearing forces during sexual intercourse and physiologic vaginal childbirth

it is clear that defects may develop weakening the endopelvic diaphragm in varying degrees from minor to extensive
it is good to realize that during pregnancy the direction of long-term pressure is from cephalad to caudal whilst during childbirth the short-term pressure is from caudal to cephalad upon this diaphragm

since it has multiple functions, like supporting the urine continence mechanism and securing the organs in their anatomic position, defects within the diaphragm will have different effects depending upon their location

the possibilities are as following: anterior, median, lateral, central and posterior; isolated or in any combination

**anterior defects**
with weakening of the urine continence support since the posterior urethra wall will “rotate backward” away from the symphysis causing vesicalization of the (proximal) urethra since fixed/adhesive anterior urethra wall
by this mechanism genuine or postrepair intrinsic stress incontinence develops

**median longitudinal defects**
depending upon its location the posterior urethra, bladder base may herniate thru this defect into the zero-pressure vagina and eventually prolapse to the outside
only if there is also concomitant weakening of the support or dorsal-directed pull on the posterior urethra wall towards the sacrum the urine continence mechanism may be involved

**central defect**
the cervix/uterus will herniate thru this defect into the vagina and then may prolapse unopposed to the outside thru the hernia-prone opening in the pelvis floor dragging the anterior vagina wall with it like intussusception
only infrequently if there is concomitant weakening of the support or dorsal-directed pull on the posterior urethra wall towards the sacrum the urine continence mechanism may be involved
normally there is full urine continence in total uterus/cervix prolapse c3 or c4 even with a urethra length of only 0.5-1 cm
however, with increased longitudinal bladder diameter, shortened urethra and narrow external urethra opening

**apical defect**
this will result in herniation of the intraperitoneal contents into the zero-pressure vagina

**posterior defects**
this will result in herniation of the rectum into the zero-pressure vagina especially when combined with perineal body defects

**lateral defects at atf**
this will result in loss of tonus of the endopelvic diaphragm and increase in the caudad/cephalad movements but not in herniation/prolapse of an organ thru this defect

**lateral defects of the fascia sheath in between the atf and atlam**
this will result in medial displacement of the atf with loss of tonus and hypermobility of the endopelvic diaphragm but not in herniation/prolapse of an organ thru this defect

**other location**
due to penetrating trauma or forceps delivery or vacuum delivery
**bb** defects with anatomic tissue loss

it is good to realize that in any obstetric urine fistula there is anatomic tissue loss of the endopelvic diaphragm/pubocervical musculofascia as well

therefore in obstetric trauma surgery one should make an effort to identify the musculo fascia defects and repair them together with the fistula

the extent and location of pressure necrosis lesions in prolonged obstructed labor may be from minimal to extensive and from one location to the other in an endless variation which makes the obstetric trauma so intriguing

**circular punched out defects**
the same size as the fistula or (slightly) bigger than the fistula

**transverse curved defects**
bigger than the fistula whereby the fistula is somewhere within this defect

**quartermircular defects**
with partial or total anatomic loss of atf and atlam and possible partial loss of levator ani muscles, obturator muscles and obturator membrane

with fistula formation and possible opening of the paravesical space

**semicircular defects**
with partial or total anatomic tissue loss of atf and atlam; and with partial tissue loss of the levator ani muscles, obturator internus muscles and obturator membrane; eventually with bare bones

with fistula formation and opening of the paravesical space

**regularly (sub)total pubocervical musculofascia loss**
regularly (sub)total fascia loss with extensive fistula formation and anterior vagina wall loss and total loss of atf and atlam and (partial/extensive) loss of levator ani muscles, obturator internus muscles and obturator membrane is found with bare bones in a so-called empty pelvis

**regularly (sub)total endopelvic diaphragm loss**
from time to time (sub)total loss of the whole diaphragm may be found with extensive soft tissue loss resulting in extensive urine/stool fistulas as cloaca; for these unfortunate women nothing can be done

however, anatomic tissue loss may also be found

due to surgery whereby tissue is excised

or due to necrotizing infections like postmeasles noma vaginae

**cc** combination of functional with anatomic defects

this combination of **aa** and **bb** is always possible and has to be checked for during the reconstructive procedure
reconstructive surgery

it is important first to identify the real (extent of the) defect(s) and then reconstruct the functional anatomy meticulously using autologous structures so that normal physiology will be ensured whilst

special attention has to be given to check that all (musculo)fascia structures are firmly (re)connected to the cervix as the centrum tendineum intrapelvinum

discussion

the endopelvic diaphragm as part of the corpus intrapelvinum is an important dynamic structure

it constitutes a real diaphragm with the cervix as its center with a small anterior median opening for the urethra and a larger posterior median opening for the rectum

separating the distal urinary tract, proximal genital tract, intraperitoneal contents and distal digestive tract (rectum) from the zero-pressure vagina

counteracting as first line the intraabdominal hydrostatic pressure due to the non-fatigue dynamic tonus of its smooth muscle component by its tonic action via baroreceptors as modulated by the autonomic nervous system; whilst the rest pressure is then dealt with by the pelvis floor structures

with immediate reactive phasic action contraction upon stretch as superimposed upon the already existing tonic action in case of sudden intraabdominal pressure rise

contributing to securing and stabilizing the pelvis organs in their variable anatomic position and as such

supporting the anatomic urine and genital continence mechanisms

defects in this diaphragm are rather common and may be due to (increased) hydrostatic pressure, shearing by vaginal childbirth, pressure necrosis during prolonged obstructed labor, penetrating trauma and necrotizing infection; as also influenced by hormonal and ageing processes

depending upon (the large variety of) the anatomic location and extent of these defects the following is possible

intrinsic stress incontinence, cervix incompetence?, urethrocele, vesicocele, uterus/cervix prolapse, enterocele and rectocele; either isolated or in combination

there is a clear correlation between genuine intrinsic urine incontinence, cystocele and cervix prolapse with a wide pubic arch of $\geq 90^\circ$ as indication of a wide pelvis

simply since the wider the pelvis the broader the span by the diaphragm and the more chance the longitudinal fibers will split/divide in the midline; with its weakest point just anteriorly from the cervix where the span is the widest
though lateral defects due to hydrostatic pressure and/or shearing at atf level and lateral defects in the narrow triangular fascia sheath between atf and atlam are possible this will not lead to herniation of the posterior bladder wall thru these defects into the vagina

at least the author has not encountered this as the cause of cystocele; the only time the author encountered a lateral defect with cystocele formation was in a patient who developed a fourth obstetric fistula after successful repair of three previous obstetric fistulas including an extensive type IIBb

in quartercircular and semicircular defects (always combined with lateral defects) with anatomic tissue loss of the endopelvic diaphragm and with fistula formation ensuring an empty bladder, another mechanism comes into play according to the natural tissue forces; besides the fact that the urethra and bladder will retract in opposite directions

which is the opposite of what one would expect

due to the balloon-like structure of the bladder with anterior bladder wall adherent/sticking to the posterior symphysis this will result in anterior and cephalad pull onto the posterior bladder (neck) wall whereby the loose endopelvic diaphragm is pulled as well and will re-attach onto the pubis bones and bilateral pelvis wall at a more anterior and cephalad level due to the natural tissue forces

actually, the saucer-like shape of the empty bladder in the normal anatomic situation is caused by the fact that the fixation of the posterior bladder wall onto the endopelvic diaphragm prevents the natural tissue forces from adapting the posterior bladder wall onto the anterior bladder wall

in identifying the endopelvic diaphragm
look for
shiny smooth muscle tissue

first edition january 2005
last edition october 2018
postscriptum

the problem with writing about the complex obstetric trauma is that there is no real scientific literature available to start with.

the available literature consists of experience reports by real obstetric fistula surgeons, political statements, rhetoric and fake information by the major ngo’s and assumptions by obstetric fistula tourists stealing the work of others and that peer reviewed

and all with fake information since the one and only cause of the obstetric fistula is poor obstetric care due to a failed system

so in prevention only this cause has to be addressed by a major combined effort of the general public, governments, who, major ngo’s and the medical professionals

and there is a trend to call things simple; however, calling something simple does not make it simple; as simply demonstrated by an average closure rate of 60-70% with an incontinence rate of 15-30% of the closed fistulas

this series is the first scientific attempt to document first the functional pelvis anatomy in the female, the mechanism of continence and incontinence, the mechanism of action of the complex obstetric trauma in its full variable extent, the real necrotic tissue loss of the different pelvis structures in a fluid endless variety, how to identify these lesions in a systematic manner and the principles of different solutions in order to reconstruct the functional pelvis anatomy, if possible

the author as a professional surgeon considers the obstetric trauma as a professional challenge which has to be solved in a professional way

though the author is trying to provide the theoretical insight as based on extensive study and anatomic findings in the living and to give the practical evidence-based solutions these are only guidelines and it is up to the reader to devise his/her own plan of action since strict protocols without insight will only lead to tricks

the strict protocols and the so-called golden standard are more to protect the doctor from litigation and criticism than that these contribute to a customized patient-oriented approach so protocols/principles/standards have to be customized

the question to be asked is: will the patient benefit from the surgical intervention

as a first edition there are quite a number of limitations, especially how to phrase one’s experience, what to include and what to exclude, is it too simple or too complicated, are the illustrations contributing etc

and already upon publication things are evolving and evolving regarding the theoretic insight and as to new operation techniques

in later editions the author will try to adapt his recommendations according to his ever evolving theoretic insight and evidence-based practical solutions

the author is confident the basics are and will be valid even if new insight and principles will develop; all over the world since the complex obstetric trauma is universal
## abbreviations

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<tr>
<th>Abbreviation</th>
<th>Definition</th>
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<tr>
<td>vvf</td>
<td>vesicovaginal fistula</td>
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<td>rvf</td>
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<td>pcmf</td>
<td>pubocervical musculofascia</td>
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<td>total abdominal hysterectomy</td>
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<td>tvh</td>
<td>total vaginal hysterectomy</td>
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abbreviations

vvf = vesicovaginal fistula
rvf = rectovaginal fistula
uvvf = urethrovesicovaginal fistula
vcvf = vesicocervicovaginal fistula
vuvf = vesicouterovaginal fistula
cx = cervix
avw = anterior vagina wall
pvw = posterior vagina wall
pcmf = pubocervical musculofascia
atf = arcus tendineus fasciae
atlam = arcus tendineus of levator ani muscle
lam = levator ani muscle
pcm = pubococcygeus muscle
ocm = obturatococcygeus muscle
iscm = (ischio)coccygeus muscle
iom = obturator internus muscle
pm = piriformis muscle
sul = sacrouterine ligament
bl = broad ligament
cl = cardinal ligament
epd = endopelvic diaphragm
ch = charrière
g = gauge
h = hegar
p = parity
sb = stillborn
cs = cesarean section
sth = subtotal hysterectomy
tah = total abdominal hysterectomy
tvh = total vaginal hysterectomy

euo = external urethra opening
iuo = internal urethra opening
uv(-junction) = urethrovesical (junction)

euo/f = distance between euo and fistula
f/c = distance between fistula and cervix
f/v = distance between fistula and vagina vault;
euo/b = distance between euo and catheter balloon
euo/bw = distance between euo and bladder wall (fundus)
a/f = distance between anus and (rectovaginal) fistula
i/v = distance between introitus and vagina vault; vagina length

pa = pubic arch
ap = anterior to posterior pelvis diameter
ar = anal reflex
gm = gastrocnemius muscle
sm = soleus muscle
at = achilles tendon

min = minute
hr = hour
wk = week
mth = month
yr = year

R = right
L = left

bladder capacity by longitudinal diameter (euo/bw minus euo/b)
small < 4 cm
moderate 5-6 cm
normal 7-12 cm
transitional 13-14 cm
increased > 15 cm
normal pelvis measurements

vagina length  10-12 cm

euo/c  6-7-8 cm

anatomic urine continence mechanism  4-5 cm
anatomic stool continence mechanism  4-5 cm

urethra length  3.5-4 cm
however, during surgery it is more in the range of 2.5-3 cm; exceptionally 5 cm

longitudinal bladder diameter (euo/bw minus euo/b)  7-12 cm

anorectum  4-5 cm

symphysis  5-6 cm broad
axis inclination  30-45° as to horizontal in the upright position

pubic arch  85-90°

atf  7.5-8 cm
inclination  25-30° as to horizontal from pubis bone to ischium spine

atlam  7-7.5 cm
inclination  25-30° as to horizontal from pubis bone to ischium spine

angle between symphysis and atf/atlam  110-125°

inter ischium spine distance  10 cm

inter ischium tuberosity distance  10-11 cm

pelvis inlet plane inclination 55-60° to horizontal from superior symphysis edge to promontory in the upright position

pelvis outlet  10-15° to horizontal from inferior symphysis to tip of coccyx  in the upright position

anterior triangle pelvis outlet from inferior symphysis to ischium tuberosity in one plane with -10 to-15° inclination to horizontal in upright position

posterior triangle pelvis outlet from ischium tuberosity to tip of coccyx in one plane with 45-50° inclination as to horizontal in upright position

angle anterior perineum/posterior perineum 55-65°/115-125°
pelvis outlet surface 75-80 sq cm

gap between levator ani ledges  25-30 sq cm

diameter recta from inferior symphysis up to tip of coccyx 9-9.5 cm; up to 10.5-11cm during delivery

perineum outlet

  spb  = symphysis to perineal body  3.5-4.5 cm
  pb height                        2 cm
  anus (+ sphincter) diameter      1.5-2 cm
  pac  = anus to coccyx bone       5-6 cm
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and as influenced by many others since the author started his medicine study in 1959
but especially by prof j m greep, prof t k a b eskes and dr med h stenkhoff