

WOMEN'S HEALTH MADE EASY

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Contents

Detailed contents	x
Preface	xxv
Acknowledgements	xxvi
About the authors	xxvii
Abbreviations	xxix

Chapter 1 Female anatomy and physiology 1

1.1 Female anatomy	2
1.2 Hormones	8
1.3 Folliculogenesis	13
1.4 The menstrual cycle	16
1.5 Puberty	21
1.6 Menopause	22
1.7 Further reading	24

Chapter 2 Menstrual disorders 27

2.1 Introduction	28
2.2 Normal menstruation	28
2.3 Heavy menstrual bleeding and other menstrual disorders	28
2.4 Non-menstrual bleeding	32
2.5 How to delay a period	32
2.6 Amenorrhoea	33
2.7 Endometriosis and adenomyosis	36
2.8 Fibroids	38
2.9 Polycystic ovary syndrome	39
2.10 Conclusion	41
2.11 Further reading	42

Chapter 3 Contraception 43

3.1 Introduction	44
3.2 The contraceptive consultation	46
3.3 Progestogen choice in contraception	61
3.4 Combined hormonal contraception	64
3.5 Progestogen-only pills	68
3.6 Injectable contraception	70
3.7 Contraceptive implant	71
3.8 Intrauterine contraception	72

3.9	Barrier methods	75
3.10	Fertility awareness	77
3.11	Male and female sterilisation	77
3.12	Emergency contraception	78
3.13	Further reading	81

Chapter 4 Sexual health 85

4.1	Introduction	86
4.2	How to talk with patients about sex	86
4.3	Supporting sexual wellbeing in our patients	87
4.4	What is sexual health?	88
4.5	Barriers to care and intersectionality	89
4.6	Providing trauma-informed care	89
4.7	Vaginal discharge	92
4.8	Vulvovaginal candidiasis	93
4.9	Bacterial vaginosis	94
4.10	Sexually transmitted infections	96
4.11	Chlamydia	99
4.12	Gonorrhoea	100
4.13	Syphilis	100
4.14	Herpes simplex virus	102
4.15	Anogenital warts	103
4.16	Human immunodeficiency virus	105
4.17	Trichomoniasis	106
4.18	Mpox	107
4.19	Urogenital commensals	108
4.20	Further reading	109

Chapter 5 Pregnancy and fertility 111

5.1	Introduction	112
5.2	Antenatal checks	112
5.3	Birth to the six weeks postnatal appointment	118
5.4	Postnatal check	119
5.5	Miscarriage	121
5.6	Nausea and vomiting in pregnancy	123
5.7	Hypertension in pregnancy and postnatal hypertension	124
5.8	Gestational diabetes	126
5.9	Anaemia in pregnancy	128
5.10	Perinatal mental health	129

5.11	Abortion care	129
5.12	Impact on sexual function	130
5.13	How to take a fertility history	131
5.14	Infertility	132
5.15	Conclusion	133
5.16	Further reading	133

Chapter 6 Menopause 135

6.1	Menopause definitions	136
6.2	Symptoms of perimenopause and menopause	136
6.3	Diagnosis of perimenopause and menopause	139
6.4	Patient assessment	142
6.5	Future health discussion	146
6.6	Management options for perimenopause and menopause	152
6.7	Genitourinary syndrome of the menopause	158
6.8	Premature ovarian insufficiency	162
6.9	Induced menopause	165
6.10	Surgical menopause	166
6.11	Menopause after cancer	168
6.12	Resources	170
6.13	Further reading	170

Chapter 7 Hormone replacement therapy 173

7.1	HRT summary	174
7.2	Types of oestrogen and progestogen used in HRT	175
7.3	Bioidentical HRT	178
7.4	Systemic HRT	179
7.5	Hormonal contraception and perimenopause	180
7.6	Tibolone	180
7.7	Initiating HRT	181
7.8	Injectable weight-loss drugs, contraception and HRT	183
7.9	HRT products and doses	184
7.10	Testing oestrogen levels	192
7.11	Contraindications to HRT	193
7.12	Managing patients taking HRT	193
7.13	Stopping HRT	194
7.14	Side-effects of HRT	195
7.15	Unscheduled bleeding on HRT	196
7.16	Benefits and risks of taking HRT	199

7.17 Prescribing HRT in certain groups	203
7.18 Sexual desire and testosterone	207
7.19 Referral to a menopause specialist	210
7.20 Resources for clinicians	210
7.21 Further reading	210

Chapter 8 Female mental health 213

8.1 Introduction to the premenstrual disorders	214
8.2 Perinatal mental health	221
8.3 Hormonal contraception and mood disorders	224
8.4 Neurodiversity in women and girls	225
8.5 Eating disorders and body dysmorphic disorder	226
8.6 Further reading	229

Chapter 9 Genitourinary issues 233

9.1 Pelvic organ prolapse	234
9.2 Urinary incontinence	237
9.3 Faecal incontinence	240
9.4 Recurrent UTI	241
9.5 Bladder pain syndrome/interstitial cystitis	244
9.6 Sexual pain/penetration disorders	245
9.7 Persistent genital arousal disorder	251
9.8 Further reading	252

Chapter 10 Vulval dermatology 255

10.1 Introduction	256
10.2 Prepubertal girls	256
10.3 Girls and women of reproductive age	258
10.4 Postmenopausal women	266
10.5 Practical tips to help manage vulval dermatoses	267
10.6 General care of the vulval skin and self-examination	269
10.7 Red flags and when to refer	269
10.8 Further reading	270

Chapter 11 Female cancer and screening 271

11.1 Introduction	272
11.2 Breast cancer and the NHS breast screening programme	272
11.3 Endometrial cancer	276
11.4 Cervical cancer and the NHS cervical cancer screening programme	277

11.5	Ovarian cancer	280
11.6	Vulval cancer	282
11.7	Vaginal cancer	284
11.8	Support after cancer	285
11.9	Conclusion	286
11.10	Further reading	286

Chapter 12 The breast 287

12.1	The breast	288
12.2	Breast symptoms	289
12.3	Benign breast conditions	291
12.4	Breast cancer	294
12.5	Breast pain	297
12.6	Nipple discharge	300
12.7	Nipple changes	301
12.8	Breastfeeding	302
12.9	Breast imaging	303
12.10	Further reading	304

Chapter 13 Legal and safeguarding 307

13.1	Consent	308
13.2	Understanding our safeguarding responsibilities	310
13.3	Sexual assault	311
13.4	Domestic violence	312
13.5	Non-fatal strangulation	315
13.6	Further reading	315
	Index	317

Detailed contents

Preface	xxv
Acknowledgements	xxvi
About the authors	xxvii
Abbreviations	xxix

Chapter 1 Female anatomy and physiology 1

1.1	Female anatomy	2
1.1.1	The pelvis	2
1.1.2	The vulva	2
1.1.3	The vagina	4
1.1.4	The cervix	5
1.1.5	The uterus	6
1.1.6	The fallopian tube	7
1.1.7	The ovary	7
1.2	Hormones	8
1.2.1	Oestrogen	8
1.2.2	Progesterone	10
1.2.3	Testosterone and androgens	11
1.2.4	Sex hormone-binding globulin	13
1.2.5	Inhibin	13
1.2.6	Growth factors	13
1.2.7	Anti-Müllerian hormone	13
1.3	Folliculogenesis	13
1.3.1	Follicular maturation	14
1.4	The menstrual cycle	16
1.4.1	The ovarian cycle	16
1.4.2	The endometrial cycle	18
1.4.3	Normal menstrual cycle length and regularity	19
1.5	Puberty	21
1.5.1	Hormonal changes	21
1.5.2	Physical changes	21
1.6	Menopause	22
1.6.1	The stages of reproductive aging	22
1.6.2	Hormonal changes in the menopause transition	23
1.6.3	Menstrual changes in perimenopause	23
1.7	Further reading	24

Chapter 2 Menstrual disorders 27

2.1	Introduction	28
2.2	Normal menstruation	28

2.3	Heavy menstrual bleeding and other menstrual disorders	28
2.3.1	Heavy menstrual bleeding	28
2.3.2	Dysmenorrhoea	30
2.3.3	Irregular menstrual cycles	31
2.3.4	Menstrual health and contraception	32
2.3.5	Premenstrual syndrome and premenstrual dysphoric disorder	32
2.4	Non-menstrual bleeding	32
2.5	How to delay a period	32
2.5.1	Methods	33
2.6	Amenorrhoea	33
2.6.1	Relative energy deficiency in sport (RED-S)	35
2.7	Endometriosis and adenomyosis	36
2.7.1	Endometriosis	36
2.7.2	Adenomyosis	38
2.8	Fibroids	38
2.9	Polycystic ovary syndrome	39
2.10	Conclusion	41
2.11	Further reading	42

Chapter 3 Contraception **43**

3.1	Introduction	44
3.1.1	Methods of contraception	44
3.1.2	UK Medical Eligibility Criteria for Contraceptive Use (UKMEC) 2025	44
3.1.3	Effectiveness of contraceptive methods	46
3.2	The contraceptive consultation	46
3.2.1	Taking a contraception history	46
3.2.2	Assessing competence – Fraser guidelines and Gillick competency	48
3.2.3	Patient information	49
3.2.4	Common questions asked by patients	49
3.2.5	Short-acting and long-acting contraception methods	50
3.2.6	Examples of questions to ask patients to help contraceptive choice	50
3.2.7	Starting a contraceptive method	51
3.2.8	Advice to include when prescribing a method of contraception	52
3.2.9	Incorrectly taken oral hormonal contraceptive pills	52
3.2.10	Contraceptive choices for women over 40 years	53
3.2.11	Overweight, obesity and contraception	53
3.2.12	Weight loss treatments and contraception	54
3.2.13	Prescribing in other groups	55
3.2.14	Non-contraceptive benefits of contraceptive methods	55
3.2.15	Contraception after having a baby	56
3.2.16	Contraception and breastfeeding	56
3.2.17	Problematic bleeding on hormonal contraception	56

3.2.18	When can contraception be stopped?	59
3.2.19	Hormone replacement therapy (HRT) and contraception	59
3.2.20	Drug interactions	60
3.2.21	Contraception for women using known teratogenic drugs	60
3.2.22	Contraception for trans men and non-binary people	61
3.2.23	Breast cancer risk and hormonal contraception	61
3.2.24	Risk of intracranial meningioma	61
3.3	Progestogen choice in contraception	61
3.3.1	Synthetic progestogen classification	62
3.3.2	Indications for use of synthetic progestogens	63
3.4	Combined hormonal contraception	64
3.4.1	Mechanism of action	64
3.4.2	Available forms of combined hormonal contraception	64
3.4.3	Types of combined oral contraceptive pill (COCP)	64
3.4.4	Advantages of combined hormonal contraception	65
3.4.5	Disadvantages of combined hormonal contraception	65
3.4.6	Starting CHC	66
3.4.7	Incorrect use of CHC (late or missed pills, ring or patch)	66
3.4.8	Vomiting and diarrhoea in women taking a COC	66
3.4.9	Seek urgent review	66
3.4.10	Tailored pill regimes	67
3.4.11	Possible side-effects of CHC and how to manage them	68
3.5	Progestogen-only pills	68
3.5.1	Mechanism of action	68
3.5.2	Available forms of POP	68
3.5.3	Advantages of POPs	69
3.5.4	Disadvantages of POPs	69
3.5.5	Starting the POP	69
3.5.6	Incorrect use of POPs	69
3.5.7	Vomiting and diarrhoea	69
3.6	Injectable contraception	70
3.6.1	Mechanism of action	70
3.6.2	Available forms of injectable contraception	70
3.6.3	Advantages of injectable contraception	70
3.6.4	Disadvantages of injectable contraception	70
3.6.5	Starting injectable contraception	71
3.6.6	Managing prolonged bleeding with DMPA	71
3.7	Contraceptive implant	71
3.7.1	Mechanism of action	71
3.7.2	Advantages of contraceptive implant	71
3.7.3	Disadvantages of contraceptive implant	71
3.7.4	Starting the IMP	72
3.7.5	Routine follow-up	72
3.7.6	Managing side-effects and bleeding	72

3.8	Intrauterine contraception	72
3.8.1	Levonorgestrel intrauterine device	72
3.8.2	Copper intrauterine device	73
3.8.3	Fitting and removal of Cu-IUD and all LNG-IUDs	74
3.8.4	IUD missing threads	75
3.8.5	Malpositioned IUD	75
3.9	Barrier methods	75
3.9.1	Mechanism of action	75
3.9.2	Types of barrier method	75
3.9.3	Condoms	75
3.9.4	Diaphragms and caps	76
3.9.5	Dental dam	76
3.9.6	Lubricants for sexual intercourse	76
3.10	Fertility awareness	77
3.11	Male and female sterilisation	77
3.12	Emergency contraception	78
3.12.1	Types of emergency contraception	78
3.12.2	Mechanism of action	78
3.12.3	Assessing pregnancy risk	78
3.12.4	Copper intrauterine device	78
3.12.5	Assessing the earliest predicted date of ovulation when considering a Cu-IUD for EC	78
3.12.6	Oral emergency contraception	79
3.12.7	Oral selective progesterone receptor modulator: UPA	79
3.12.8	Oral progestogen-only EC: LNG-EC	79
3.12.9	After taking emergency contraception	80
3.13	Further reading	81

Chapter 4 Sexual health 85

4.1	Introduction	86
4.2	How to talk with patients about sex	86
4.2.1	Further training in psychosexual medicine	87
4.3	Supporting sexual wellbeing in our patients	87
4.4	What is sexual health?	88
4.4.1	Where does pleasure fit in?	88
4.5	Barriers to care and intersectionality	89
4.5.1	What is intersectionality?	89
4.6	Providing trauma-informed care	89
4.6.1	What do we mean by trauma?	89
4.6.2	How can trauma affect us?	90
4.6.3	How to conduct a trauma-informed examination	91
4.7	Vaginal discharge	92
4.7.1	Normal physiological discharge	92
4.7.2	Vaginal discharge during the menstrual cycle	92

4.7.3	Vaginal discharge with contraceptives, and during pregnancy and menopause	92
4.7.4	When to investigate vaginal discharge and test for STIs	92
4.8	Vulvovaginal candidiasis	93
4.8.1	What is vulvovaginal candidiasis and who does it affect?	93
4.8.2	How do we diagnose vulvovaginal candidiasis?	93
4.8.3	Recurrent vulvovaginal candidiasis	93
4.8.4	Self-care advice for vulvovaginal candidiasis	93
4.8.5	Treatment of vulvovaginal candidiasis	94
4.8.6	Treatment of recurrent vulvovaginal candidiasis	94
4.8.7	Treatment of vulvovaginal candidiasis in pregnancy and breastfeeding	94
4.9	Bacterial vaginosis	94
4.9.1	What is bacterial vaginosis and who does it affect?	94
4.9.2	How do we diagnose bacterial vaginosis?	95
4.9.3	How do we treat bacterial vaginosis?	95
4.9.4	Bacterial vaginosis in pregnancy	95
4.10	Sexually transmitted infections	96
4.10.1	Identification – who is at risk of STIs?	96
4.10.2	How do we know who needs to be tested and what for?	96
4.11	Chlamydia	99
4.11.1	What is chlamydia and who is at risk?	99
4.11.2	How do we diagnose chlamydia?	99
4.11.3	How do we treat chlamydia?	99
4.12	Gonorrhoea	100
4.12.1	What is gonorrhoea and who is at risk?	100
4.12.2	How do we diagnose and treat gonorrhoea?	100
4.13	Syphilis	100
4.13.1	What is syphilis and who is at risk?	100
4.13.2	How do we diagnose and treat syphilis?	101
4.14	Herpes simplex virus	102
4.14.1	What is herpes simplex virus and who is at risk?	102
4.14.2	How do we diagnose and treat HSV?	102
4.14.3	What about HSV infection in pregnancy or immunocompromised individuals?	103
4.14.4	What about recurrent HSV infection?	103
4.15	Anogenital warts	103
4.15.1	What are anogenital warts and who is at risk?	103
4.15.2	How do we diagnose and treat anogenital warts?	104
4.15.3	What about HPV in pregnancy/immunocompromised individuals?	105
4.16	Human immunodeficiency virus	105
4.16.1	What is HIV and who is at risk?	105
4.16.2	How do we diagnose and treat HIV?	105
4.16.3	HIV prophylaxis options	106

4.17	Trichomoniasis	106
4.17.1	What is trichomoniasis and who is at risk?	106
4.17.2	How do we diagnose and treat trichomoniasis?	107
4.18	Mpox	107
4.19	Urogenital commensals	108
4.19.1	<i>Ureaplasma urealyticum</i> and <i>U. parvum</i>	108
4.19.2	Mycoplasma bacteria	108
4.20	Further reading	109

Chapter 5 Pregnancy and fertility 111

5.1	Introduction	112
5.2	Antenatal checks	112
5.2.1	Pre-conception care	113
5.2.2	First antenatal appointment	115
5.2.3	First antenatal booking visit	115
5.2.4	First ultrasound scan	116
5.2.5	16 weeks antenatal appointment	116
5.2.6	Second ultrasound scan	116
5.2.7	25 weeks antenatal appointment	116
5.2.8	28 weeks antenatal appointment	116
5.2.9	31 weeks antenatal appointment	117
5.2.10	34 weeks antenatal appointment	117
5.2.11	36 weeks antenatal appointment	117
5.2.12	38 weeks antenatal appointment	117
5.2.13	40 weeks antenatal appointment	118
5.2.14	41 weeks antenatal appointment	118
5.3	Birth to the six weeks postnatal appointment	118
5.4	Postnatal check	119
5.4.1	Review of pregnancy / birth experience	119
5.4.2	Check for physical recovery	119
5.4.3	Cervical screening, contraception and sexual health	120
5.4.4	Assessment of perinatal mental health	120
5.4.5	Infant feeding	120
5.5	Miscarriage	121
5.5.1	Types or stages of miscarriage	121
5.5.2	Role of the healthcare professional in managing miscarriages	122
5.5.3	Ectopic pregnancy	122
5.6	Nausea and vomiting in pregnancy	123
5.6.1	Prevalence	123
5.6.2	Management	123

5.7	Hypertension in pregnancy and postnatal hypertension	124
5.7.1	Prevalence	124
5.7.2	Risk factors for hypertension and pre-eclampsia	124
5.7.3	Symptoms	125
5.7.4	Assessment	125
5.7.5	Management	125
5.7.6	Postnatal hypertension	126
5.8	Gestational diabetes	126
5.8.1	Overview	126
5.8.2	Risk factors for gestational diabetes	127
5.8.3	Screening	127
5.8.4	Diagnosis	127
5.8.5	Management	127
5.8.6	Managing gestational diabetes postnatally	128
5.9	Anaemia in pregnancy	128
5.9.1	Prevalence	128
5.9.2	Causes	128
5.9.3	Symptoms	128
5.9.4	Screening	128
5.9.5	Treatment	128
5.10	Perinatal mental health	129
5.11	Abortion care	129
5.11.1	Legal context	129
5.11.2	Role of the healthcare professional	129
5.11.3	Methods	129
5.12	Impact on sexual function	130
5.12.1	Common concerns	130
5.12.2	Causes	130
5.12.3	Management	130
5.13	How to take a fertility history	131
5.13.1	Importance of the history	131
5.13.2	Key components of history-taking	131
5.14	Infertility	132
5.14.1	Initial primary care exam and investigations	132
5.14.2	What advice to give to couples trying to conceive?	132
5.14.3	Referral criteria	133
5.15	Conclusion	133
5.16	Further reading	133
Chapter 6 Menopause		135
6.1	Menopause definitions	136
6.2	Symptoms of perimenopause and menopause	136

6.2.1	Main symptoms	137
6.2.2	Other symptoms reported by women	138
6.2.3	Menopause in ethnic minority women	138
6.3	Diagnosis of perimenopause and menopause	139
6.3.1	Women over the age of 45	139
6.3.2	Women under the age of 45	140
6.3.3	Follicle-stimulating hormone testing	141
6.3.4	Use of serum FSH measurement in assessing requirement for hormonal contraception	141
6.3.5	Contraception in perimenopause	141
6.4	Patient assessment	142
6.4.1	Patient assessment	142
6.4.2	Example of a symptom questionnaire chart	144
6.4.3	Assessment of symptoms of genitourinary syndrome of the menopause	145
6.5	Future health discussion	146
6.5.1	Metabolic health	146
6.5.2	Coronary heart disease	146
6.5.3	Osteoporosis	147
6.5.4	Assessment of fragility fracture risk	148
6.5.5	Dementia	150
6.6	Management options for perimenopause and menopause	152
6.6.1	Lifestyle	152
6.6.2	Hormone replacement therapy	153
6.6.3	Combined hormonal contraception	154
6.6.4	Progestogen-only contraception	154
6.6.5	Unregulated hormonal preparations	154
6.6.6	Cognitive behavioural therapy	154
6.6.7	Non-hormonal prescribable medications for the relief of vasomotor symptoms	155
6.6.8	Complementary and alternative therapies	156
6.6.9	Menopause and work	157
6.6.10	Support for specific symptoms	157
6.7	Genitourinary syndrome of the menopause	158
6.7.1	Localised vaginal oestrogen products	160
6.7.2	Use of vaginal oestrogen in women with a personal history of breast cancer	161
6.8	Premature ovarian insufficiency	162
6.8.1	Causes of POI	163
6.8.2	When to suspect POI	163
6.8.3	Criteria for diagnosis	163
6.8.4	Risks of POI	163
6.8.5	Management and monitoring of POI	164
6.8.6	Hormone therapy in women with POI	164
6.8.7	Fertility and contraception	165
6.9	Induced menopause	165
6.9.1	Reasons for a medically-induced menopause	166

6.10	Surgical menopause	166
6.10.1	Indications	167
6.10.2	Risks and benefits of surgical menopause	167
6.10.3	Management	167
6.10.4	HRT after surgical menopause for endometriosis	168
6.11	Menopause after cancer	168
6.11.1	Managing menopause after cancer	169
6.11.2	Patient support	169
6.11.3	Clinical resources	170
6.12	Resources	170
6.12.1	Resources for clinicians	170
6.12.2	Resources for women	170
6.13	Further reading	170

Chapter 7 Hormone replacement therapy 173

7.1	HRT summary	174
7.2	Types of oestrogen and progestogen used in HRT	175
7.2.1	Oestrogen	175
7.2.2	Progestogen	176
7.2.3	Choice of progestogen for endometrial protection as part of HRT	178
7.3	Bioidentical HRT	178
7.4	Systemic HRT	179
7.4.1	Types of systemic HRT	179
7.4.2	When to change from sequential to continuous combined HRT?	180
7.5	Hormonal contraception and perimenopause	180
7.6	Tibolone	180
7.7	Initiating HRT	181
7.7.1	Oral or transdermal oestrogen preparations and VTE risk	182
7.8	Injectable weight-loss drugs, contraception and HRT	183
7.8.1	Contraception and GLP-1 agonist use	183
7.8.2	HRT and GLP-1 agonist use	184
7.9	HRT products and doses	184
7.9.1	Oestrogen-only products	186
7.9.2	Adjuvant progestogen preparations and recommended doses (to be used cyclically or continuously, alongside an oestrogen product) in HRT	187
7.9.3	Sequential combined oestrogen and progestogen products	189
7.9.4	Continuous combined oestrogen and progestogen products	189
7.9.5	HRT doses	190
7.9.6	High-dose oestrogen	191
7.10	Testing oestrogen levels	192

7.11	Contraindications to HRT	193
7.12	Managing patients taking HRT	193
7.12.1	How long does it take for HRT to work?	194
7.12.2	Poor response to HRT	194
7.13	Stopping HRT	194
7.14	Side-effects of HRT	195
7.15	Unscheduled bleeding on HRT	196
7.15.1	Assessment of women who have unscheduled bleeding on HRT	197
7.15.2	Managing HRT after unscheduled bleeding	198
7.15.3	Adjusting HRT to reduce episodes of unscheduled bleeding	198
7.16	Benefits and risks of taking HRT	199
7.16.1	Benefits of HRT	199
7.16.2	Risks of HRT	200
7.16.3	Individualising HRT for metabolic health	203
7.17	Prescribing HRT in certain groups	203
7.17.1	Migraine	203
7.17.2	Women with a personal history of VTE or an inherited thrombophilic disorder	204
7.17.3	Women with a personal history of coronary disease or stroke	204
7.17.4	Women over 60, or more than 10 years after menopause	204
7.17.5	History of fibroid	205
7.17.6	Epilepsy	205
7.17.7	Thyroid disorder	205
7.17.8	Induced menopause in women with endometriosis	205
7.17.9	PMS/PMDD/PME	205
7.17.10	HRT and surgery	206
7.17.11	HRT after risk-reducing surgery	206
7.17.12	Early menopause (40–45 years)	206
7.17.13	Menopause after cancer	206
7.17.14	Learning difficulties	207
7.17.15	Transgender gender-affirming hormone therapy: past use	207
7.18	Sexual desire and testosterone	207
7.18.1	Talking about libido and sex	207
7.18.2	When is testosterone indicated?	208
7.18.3	Investigations	208
7.18.4	Prescribing testosterone	208
7.18.5	Possible side-effects of testosterone	209
7.18.6	Testosterone treatment options	209
7.18.7	Ongoing monitoring and follow-up	209
7.18.8	Patient information	210
7.19	Referral to a menopause specialist	210
7.20	Resources for clinicians	210
7.21	Further reading	210

Chapter 8 Female mental health 213

8.1	Introduction to the premenstrual disorders	214
8.1.1	What are premenstrual disorders and who is affected?	214
8.1.2	What causes the premenstrual disorders?	214
8.1.3	Making the diagnosis	215
8.1.4	Classification of premenstrual disorders	215
8.1.5	Premenstrual dysphoric disorder	216
8.1.6	Management of the premenstrual disorders	218
8.1.7	Useful resources for professionals and patients	221
8.2	Perinatal mental health	221
8.2.1	Definition and risk factors	221
8.2.2	Prevalence and impact	222
8.2.3	Prognosis	222
8.2.4	Diagnosis	222
8.2.5	Managing perinatal depression	223
8.2.6	Postpartum psychosis	224
8.2.7	Bipolar postpartum depression	224
8.2.8	Perinatal obsessive–compulsive disorder	224
8.3	Hormonal contraception and mood disorders	224
8.4	Neurodiversity in women and girls	225
8.4.1	What is neurodiversity and who is affected?	225
8.5	Eating disorders and body dysmorphic disorder	226
8.5.1	Introduction	226
8.5.2	How can we diagnose eating disorders?	227
8.6	Further reading	229

Chapter 9 Genitourinary issues 233

9.1	Pelvic organ prolapse	234
9.1.1	What is pelvic organ prolapse and who is affected?	234
9.1.2	Types and stages of pelvic organ prolapse	234
9.1.3	Preventing prolapse	235
9.1.4	Treating prolapse	235
9.2	Urinary incontinence	237
9.2.1	What is urinary incontinence and who is affected?	237
9.2.2	How do we diagnose and assess urinary continence issues?	238
9.2.3	Managing urinary incontinence: when to refer?	238
9.2.4	General management of urinary incontinence within primary care	239
9.2.5	Management of stress urinary incontinence	239
9.2.6	Management of urgency incontinence / overactive bladder	239
9.2.7	Management of mixed urinary incontinence	239
9.3	Faecal incontinence	240
9.3.1	Assessment of faecal incontinence	240
9.3.2	Management of faecal incontinence	241

9.4	Recurrent UTI	241
9.4.1	Definitions	241
9.4.2	Aetiology of recurrent UTI	242
9.4.3	What puts some women at risk for recurrent UTI?	242
9.4.4	Diagnosing recurrent UTI	242
9.4.5	Treatment and prevention of recurrent UTI	242
9.4.6	Chronic embedded UTI	243
9.5	Bladder pain syndrome / interstitial cystitis	244
9.5.1	Aetiology of bladder pain syndrome / interstitial cystitis	244
9.5.2	Diagnosis of bladder pain syndrome / interstitial cystitis	244
9.5.3	Management of bladder pain syndrome / interstitial cystitis	244
9.6	Sexual pain / penetration disorders	245
9.6.1	Vulvar pain and vulvodynia	245
9.6.2	Vaginismus	247
9.6.3	Pudendal neuralgia	250
9.7	Persistent genital arousal disorder	251
9.8	Further reading	252

Chapter 10 Vulval dermatology 255

10.1	Introduction	256
10.2	Prepubertal girls	256
10.2.1	Inflammatory and dermatitis-related conditions	256
10.2.2	Infectious dermatoses	257
10.2.3	Trauma and other causes	257
10.3	Girls and women of reproductive age	258
10.3.1	Normal vulval skin variants	258
10.3.2	Common vulval dermatoses	258
10.3.3	Female genital mutilation	266
10.3.4	Vulval pain syndromes	266
10.4	Postmenopausal women	266
10.4.1	Common vulval dermatoses seen in postmenopausal women	267
10.4.2	Vulval changes during menopause	267
10.5	Practical tips to help manage vulval dermatoses	267
10.6	General care of the vulval skin and self-examination	269
10.7	Red flags and when to refer	269
10.8	Further reading	270

Chapter 11 Female cancer and screening 271

11.1	Introduction	272
11.2	Breast cancer and the NHS breast screening programme	272
11.2.1	Epidemiology	272
11.2.2	Risk factors	272

11.2.3	Clinical presentation	273
11.2.4	Diagnosis	273
11.2.5	Treatment	274
11.2.6	UK screening recommendations (NHS breast screening programme)	275
11.2.7	The role of the clinician	275
11.3	Endometrial cancer	276
11.3.1	Epidemiology	276
11.3.2	Risk factors	276
11.3.3	Clinical presentation	276
11.3.4	Diagnosis	276
11.3.5	Treatment	277
11.3.6	UK screening recommendations	277
11.3.7	The role of the clinician	277
11.4	Cervical cancer and the NHS cervical cancer screening programme	277
11.4.1	Epidemiology	277
11.4.2	Risk factors	277
11.4.3	Clinical presentation	278
11.4.4	Diagnosis	278
11.4.5	Treatment	279
11.4.6	UK screening recommendations (NHS cervical screening programme)	279
11.4.7	The role of the clinician	280
11.5	Ovarian cancer	280
11.5.1	Epidemiology	280
11.5.2	Risk factors	281
11.5.3	Clinical presentation	281
11.5.4	Diagnosis	282
11.5.5	Treatment	282
11.5.6	UK screening recommendations	282
11.5.7	The role of the clinician	282
11.6	Vulval cancer	282
11.6.1	Epidemiology	282
11.6.2	Risk factors	283
11.6.3	Clinical presentation	283
11.6.4	Diagnosis	283
11.6.5	Treatment	283
11.6.6	UK screening recommendations	283
11.6.7	The role of the clinician	284
11.7	Vaginal cancer	284
11.7.1	Epidemiology	284
11.7.2	Risk factors	284
11.7.3	Clinical presentation	284
11.7.4	Diagnosis	284
11.7.5	Treatment	285
11.7.6	The role of the clinician	285

11.8	Support after cancer	285
11.9	Conclusion	286
11.10	Further reading	286

Chapter 12 The breast **287**

12.1	The breast	288
12.1.1	Breast size and density	289
12.1.2	Breast awareness	289
12.2	Breast symptoms	289
12.2.1	History-taking	290
12.2.2	Risk factors for breast cancer	290
12.2.3	Breast examination	290
12.3	Benign breast conditions	291
12.3.1	Benign breast swelling and tenderness	291
12.3.2	Breast cysts	291
12.3.3	Fibroadenoma	291
12.3.4	Fat necrosis	292
12.3.5	Benign phyllodes	292
12.3.6	Intraductal papilloma	292
12.3.7	Atypical hyperplasia	292
12.3.8	Sclerosing adenosis	292
12.3.9	Duct ectasia	292
12.3.10	Periductal mastitis	293
12.3.11	Granulomatous mastitis	293
12.3.12	Infection – mastitis	293
12.3.13	Skin lesions on the breast	293
12.3.14	Breast nodularity	294
12.4	Breast cancer	294
12.4.1	Signs and symptoms suggestive of breast cancer	294
12.4.2	Referral	295
12.4.3	Family history of breast cancer	295
12.4.4	Referral from primary care for family history of breast cancer	296
12.4.5	Risk of breast cancer	296
12.4.6	Inherited genetic mutations increasing risk of breast cancer	297
12.5	Breast pain	297
12.5.1	What to include in the history and examination	298
12.5.2	Types of breast pain	298
12.6	Nipple discharge	300
12.6.1	Taking a history	300
12.6.2	Differential diagnosis	300
12.6.3	Management of nipple discharge	300
12.7	Nipple changes	301
12.7.1	Taking a history	301

12.7.2	Nipple inversion	302
12.7.3	Nipple retraction	302
12.7.4	Nipple eczema	302
12.7.5	Paget's disease of the breast	302
12.7.6	Referral	302
12.8	Breastfeeding	302
12.8.1	Lactation physiology	302
12.8.2	Breast milk composition	303
12.8.3	Breastfeeding problems	303
12.9	Breast imaging	303
12.9.1	Mammogram	303
12.9.2	Ultrasound	304
12.9.3	Magnetic resonance imaging	304
12.9.4	Clinician resources	304
12.10	Further reading	304
Chapter 13 Legal and safeguarding		307
13.1	Consent	308
13.1.1	What is consent?	308
13.1.2	What is required for consent to be given?	308
13.1.3	Young people and consent	308
13.1.4	Learning disability, consent and women's health	309
13.2	Understanding our safeguarding responsibilities	310
13.2.1	Specific safeguarding concerns: female genital mutilation	310
13.3	Sexual assault	311
13.3.1	Responding to a disclosure of a recent sexual assault	312
13.3.2	Supporting women after sexual assault	312
13.3.3	Helpful resources	312
13.4	Domestic violence	312
13.4.1	Who is most at risk?	313
13.4.2	What can we do if a woman discloses that she is experiencing abuse?	313
13.4.3	Multi-agency risk assessment conferences (MARACs)	314
13.4.4	Helpful resources	314
13.5	Non-fatal strangulation	315
13.6	Further reading	315
	Index	317

Preface

Women's health is often described as "complex". It is probably more accurate to say it has been made so by decades of under-research, under-teaching, and historical bias within medicine.

For too long, women's symptoms have been minimised, normalised, or attributed to psychology. At the same time, clinicians have been expected to manage high-impact, intimate areas of care with limited formal training, patchy evidence and inconsistent guidance. This has created significant and systemic challenges in women's health: shown through delayed diagnoses, fragmented care and a growing sense of loss of trust in healthcare.

Women's Health Made Easy was written in response to these gaps. Invited to develop this text through the Primary Care Women's Health Society, we set out to create a practical, clinically useful resource that was grounded in real consultations. Drawing on our combined experience as GPs, menopause specialists and sexologists, we focus on areas historically underrepresented in training, including sexual health, vulval conditions, menstrual disorders, fertility and menopause, alongside communication, safeguarding, perinatal mental health and the interaction between hormonal and psychological wellbeing.

Improving women's healthcare requires better education, better support, and access to safe, pragmatic advice based on good evidence. This book is one contribution towards that change.

Note: throughout the text we use the words 'woman' and 'women', but the information and advice in these pages is intended to include all those assigned female at birth.

*Carys Sonnenberg
Angela Wright
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Abbreviations

ACE	angiotensin-converting enzyme	DMPA	depot medroxyprogesterone acetate
ADHD	attention deficit hyperactivity disorder	DRSP	drosiprenone
AIDS	acquired immune deficiency syndrome	DSG	desogestrel
AN	anorexia nervosa	DSM	<i>Diagnostic and Statistical Manual of Mental Disorders</i>
ARB	angiotensin II receptor blocker	DYG	dydrogesterone
ART	antiretroviral therapy	EC	emergency contraception
ASD	autism spectrum disorder	ECG	electrocardiogram
BASHH	British Association for Sexual Health and HIV	ED	everyday
BDD	body dysmorphic disorder	EE	ethinylestradiol
BMD	bone mineral density	EPAU	Early Pregnancy Assessment Unit
BMI	body mass index	EPO	evening primrose oil
BMS	British Menopause Society	FAI	free androgen index
BP	blood pressure	FGM	female genital mutilation
BPS	bladder pain syndrome	FRAX	fracture risk assessment tool
BSO	bilateral salpingo-oophorectomy	FSH	follicle-stimulating hormone
BV	bacterial vaginosis	GAG	glycosaminoglycan
CAM	complementary and alternative medicine	GBMSM	gay and bisexual and men who have sex with men
CAP	complementary and alternative practices	GMC	General Medical Council
CBT	cognitive behavioural therapy	GnRH	gonadotrophin-releasing hormone
CBTi	CBT for insomnia	GPD	genito-pelvic dysthesia
ccHRT	continuous combined HRT	GSM	genitourinary syndrome of the menopause
CEE	conjugated equine oestrogen	GUM	genitourinary medicine
CHC	combined hormonal contraception	HDL	high-density lipoprotein
CHD	coronary heart disease	HER2	human epidermal growth receptor 2
CIN	cervical intraepithelial neoplasia	HFI	hormone-free interval
COC	combined oral contraceptive	HIV	human immunodeficiency virus
COCP	combined oral contraceptive pill	HMB	heavy menstrual bleeding
CoSRH	College of Sexual & Reproductive Healthcare	HPO	hypothalamic–pituitary–ovarian
COSRT	College of Sexual and Relationship Therapists	HPV	human papillomavirus
CTP	contraceptive transdermal patch	HRT	hormone replacement therapy
Cu-IUD	copper IUD	HSV	herpes simplex virus
CUV	cliterourethrovaginal	HVS	high vaginal swab
CVD	cardiovascular disease	IC	interstitial cystitis
CVR	contraceptive vaginal ring	ICD	<i>International Classification of Diseases</i>
DCIS	ductal carcinoma <i>in situ</i>	IDVA	independent domestic violence advisor
DES	diethylstilboestrol	IGF	insulin-like growth factor
DEXA	dual-energy X-ray absorptiometry	IMB	intermenstrual bleeding
DHEA	dehydroepiandrosterone	IMP	implant
DHT	dihydrotestosterone	IMS	International Menopause Society
		IPM	Institute of Psychosexual Medicine
		ISPMD	International Society for Premenstrual Disorders

IUD	intrauterine device	PGAD	persistent genital arousal disorder
IV	intravenous	PID	pelvic inflammatory disease
IVF	<i>in vitro</i> fertilisation	PMB	postmenopausal bleeding
LARC	long-acting reversible contraceptive	PMD	premenstrual disorder
LCIS	lobular carcinoma <i>in situ</i>	PMDD	premenstrual dysphoric disorder
LFT	liver function test	PMS	premenstrual syndrome
LH	luteinising hormone	PN	pubertal neuralgia
LMP	last menstrual period	PND	postnatal depression
LNG	levonorgestrel	POI	premature ovarian insufficiency
LNG-EC	levonorgestrel emergency contraception	POP	progestogen-only pill
LNG-IUD	levonorgestrel intrauterine device	POP-Q	pelvic organ prolapse quantification
MARAC	multi-agency risk assessment conference	POTS	postural orthostatic tachycardia syndrome
MC&S	microscopy, culture and sensitivities	PrEP	pre-exposure prophylaxis
Mgen	<i>Mycoplasma genitalium</i>	RCOG	Royal College of Obstetricians & Gynaecologists
MHRA	Medicines and Healthcare products Regulatory Agency	RCT	randomised controlled trial
MHT	menopause hormone therapy	RED-S	relative energy deficiency in sport
MI	myocardial infarction	SARC	Sexual Assault Referral Centre
MP	micronised progesterone	SCC	squamous cell carcinoma
MPA	medroxyprogesterone acetate	SHBG	sex hormone-binding globulin
MRI	magnetic resonance imaging	sHRT	sequential combined HRT
MS	multiple sclerosis	SNRI	serotonin–noradrenaline reuptake inhibitor
MSM	men who have sex with men	SPC	Summary of Product Characteristics
MSU	mid-stream urine	SSRI	selective serotonin reuptake inhibitor
NAPS	National Association for Premenstrual Syndromes	STI	sexually transmitted infection
NET	norethisterone NFS non-fatal strangulation	T2DM	type 2 diabetes mellitus
NFS	non-fatal strangulation	TCA	tricyclic antidepressant
NOGG	National Osteoporosis Guidelines Group	TENS	transcutaneous nerve stimulation
NSAID	non-steroidal anti-inflammatory drug	TOP	termination of pregnancy
OAB	overactive bladder	TV	trichomonas vaginalis
OCD	obsessive–compulsive disorder	U&Es	urea and electrolytes
OGTT	oral glucose tolerance test	UCP	urgent care pathway
OSA	obstructive sleep apnoea	UFED	unspecified feeding or eating disorder
OSFED	other specified feeding or eating disorder	UK NSC	UK National Screening Committee
OTC	over the counter	UKMEC	UK Medical Eligibility Criteria for Contraceptive Use
PCB	postcoital bleeding	UPA	ulipristal acetate
PCOS	polycystic ovary syndrome	USC	urgent suspected cancer
PCR	polymerase chain reaction	UTI	urinary tract infection
PCWHS	Primary Care Women’s Health Society	UUI	urge urinary incontinence
PEP	post-exposure prophylaxis	VIN	vulval intraepithelial neoplasia
PFMT	pelvic floor muscle training	VMS	vasomotor symptom
		VTE	venous thromboembolism
		WHI	Women’s Health Initiative
		WHO	World Health Organization

Chapter 8

Female mental health

8.1	Introduction to the premenstrual disorders.....	214
8.2	Perinatal mental health.....	221
8.3	Hormonal contraception and mood disorders.....	224
8.4	Neurodiversity in women and girls.....	225
8.5	Eating disorders and body dysmorphic disorder.....	226
8.6	Further reading.....	229

8.1 Introduction to the premenstrual disorders

8.1.1 What are premenstrual disorders and who is affected?

- Premenstrual disorders (PMDs) is an umbrella term for several distinct conditions, including the widely-recognised premenstrual syndrome (PMS) and the most severe form of premenstrual dysphoric disorder, PMDD.
- Most women are aware of physical and/or psychological changes that occur during their menstrual cycle. The exact nature of the symptoms experienced physically and mentally varies.
- Despite the prevalence of the premenstrual disorders, they remain poorly recognised and undertreated.
- The impact of premenstrual disorders varies in severity.
 - Severe premenstrual disorders (including PMDD) have an enormous impact on women's physical, social, psychological and economic wellbeing.
 - In contrast, women who experience minor, transient premenstrual symptoms that do not impair their activities or affect their quality of life are described as having physiological premenstrual 'symptoms' rather than premenstrual 'syndrome'.
- A recent meta-analysis suggests that PMS affects approximately half of women of reproductive age worldwide.
- PMDD is thought to affect between 3.2 and 7.7% of women of reproductive age worldwide.
- Women with PMDD are almost seven times more likely to attempt suicide and almost four times as likely to exhibit suicidal ideation than women without the condition.
- Women with PMS are also at higher risk of suicidal ideation, but not suicide attempts.
- The key to effective management of the premenstrual conditions lies in symptom recognition and accurate diagnosis.
- **Many women who are susceptible to hormone-related mood change will have vulnerability across their reproductive lives.**
- Susceptibility to PMDs and other forms of reproductive depression seems more prevalent in women who are neurodivergent, e.g. with autistic spectrum disorders or ADHD. History of exposure to trauma is also more common in women with PMDs. There is also diagnostic overlap with joint hypermobility and autonomic dysfunction.

8.1.2 What causes the premenstrual disorders?

- Understanding the aetiology of the PMDs remains an ongoing area of research.
- It is clear that ovulation, and therefore the presence of luteal phase hormones, is a key factor in the premenstrual disorders, as symptoms of PMDs do not occur prior to menarche, during pregnancy or after menopause.
- The exact cause remains uncertain, however. **Research shows hormone levels are not distinguishable between women with and without PMDs** – showing it is not hormones per se that are responsible for the condition, rather that individuals with PMD have an atypical response to them.
- Overlapping genetic and environmental factors seem to feed into a state of neurobiological vulnerability to PMDs. Research is focused in four main areas:
 - **Genetic susceptibility**
 - Studies suggest some heritability in PMDD.
 - **Dysregulation in the serotonergic system**
 - Changing hormone levels may impact the serotonin system. Depletion of tryptophan, the main precursor of serotonin, has been shown to worsen premenstrual symptoms. The efficacy of SSRIs in treatment of the PMDs also supports this theory.
 - Oestrogen and progesterone levels also affect the dopamine system.
 - **The effect of progesterone and its metabolite, allopregnanolone, on the GABAergic system**

- In people with PMDs there appears to be increased sensitivity to changes in allopregnanolone levels.
- Allopregnanolone works as an agonist at the GABA-A receptor to enhance the neurotransmitter's calming effect on mood.
- SSRIs may also alter allopregnanolone levels, which may explain how they can be effective treatment in PMD.
- **Stress and inflammation**
 - Stress seems to have a role through amplifying sympathetic activity.
 - **There may also be an exaggerated immune–inflammatory response.**

8.1.3 Making the diagnosis

- **In order to diagnose a premenstrual disorder, we must demonstrate the following:**
 - Cyclicity with respect to luteal phase.
 - Relief of symptoms after onset of menses, with a symptom-free period.
 - Impact on daily functioning.
 - Presence of symptoms over at least two consecutive cycles (useful menstrual diaries for patients can be downloaded from www.IAPMD.org).
- Symptom persistence and severity can fluctuate: one study showed that only 36% of women who met the diagnostic criteria for PMS continued to meet the diagnostic criteria one year later.
- Postmenopausal women with previous PMDs may experience recurrence of psychological and physical symptoms when they receive progestogen therapy.
- Research indicates that reproductive steroids affect virtually every system implicated in the pathophysiology of depression. **Women who are susceptible to hormone-induced mood change may also show mood change with pregnancy, the postnatal period, infertility treatment, perimenopause and menopause, and when exogenous hormones are prescribed as contraception or HRT.**
- This susceptibility to sex-steroid-related mood change is widely recognised as creating a 'window of increased vulnerability' to mental health conditions during the reproductive years.

8.1.4 Classification of premenstrual disorders

- Historically, the nomenclature and definitions for the premenstrual disorders have been varied and confusing.
- The International Society for Premenstrual Disorders (ISPMD) produced the first consensus on definitions and diagnostic criteria. This now underpins the definitions in the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition (DSM-5) and the *International Classification of Diseases*, 11th revision (ICD-11). ISPMD also developed the clinical standards for managing PMDD which underpin the Treatment Guidelines released by the Royal College of Obstetricians & Gynaecologists (RCOG) in 2013.
- The ISPMD defined premenstrual disorders as core PMDs (associated with ovulatory cycles) or variant PMDs (see also *Table 8.1*).
 - **Core premenstrual disorders** (incorporating premenstrual syndrome and PMDD)
 - Symptoms occur regularly in ovulating women.
 - They are generally present during the luteal phase.
 - They generally resolve by the end of menstruation.
 - A key diagnostic point is that a symptom-free interval is present.
 - **Variant premenstrual disorders** include everything that does not meet the criteria for the core PMDs. There are four subtypes:
 - **Premenstrual exacerbation (PME):** where there is a physical condition (e.g. asthma, migraine, epilepsy) and/or a psychological condition (e.g. depression, anxiety, eating disorders or obsessive–compulsive disorder, OCD) which is present throughout the month but worsens cyclically (usually in the luteal phase).

Table 8.1: Differentiating between core and variant PMD

Core PMD	
Subclassified according to nature of symptoms:	Symptoms occur in ovulatory cycle
• Predominantly physical	Symptoms can be physical or psychological
• Predominantly emotional	Symptoms absent after menstruation and before ovulation
• Mixed	Symptoms must be prospectively rated for at least 2 cycles
	Symptoms must cause functional impairment
Variant PMD	
Premenstrual exacerbation	Exacerbation of an underlying somatic (e.g. asthma, migraine) or psychological disorder that is present throughout the month but worsens in the luteal phase
Non-ovulatory PMD	Poorly understood and rare; symptoms arise from follicular activity of the ovary
Progestogen-induced PMD	PMD symptoms arise from exogenous sources of progestogen in the COCP or HRT
PMD with absent menstruation	PMD arises from cyclical ovarian activity even though menstruation has been suppressed – e.g. in women who have had endometrial ablation, have a 52mg LNG-IUD or have had a hysterectomy

- **PMD with absent menstruation:** this may happen when amenorrhoea has been induced, e.g. following LNG-IUD insertion, endometrial ablation or hysterectomy with conservation of the ovaries. Symptoms are thought to arise from cyclical ovarian activity despite absence of ovulation.
- **Progestogen-induced PMD:** in susceptible women, the exogenous hormones in sequential HRT or hormonal contraceptives can induce symptoms. Progestogen-only contraceptives and continuous HRT can also cause PMD-like symptoms but as these are non-cyclical, they are not included in this definition and are considered as adverse effects of continuous hormonal therapies (colloquially this is often referred to as 'progestogen sensitivity').
- **Non-ovulatory PMD:** this disorder is poorly understood but it is thought that in some women, follicular activity can precipitate symptoms even if ovulation does not occur.

8.1.5 Premenstrual dysphoric disorder

- It was only in 2013 that PMDD was clearly defined and included as a distinct diagnosis in the DSM-5.
- In 2019 the ICD-11 also included PMDD as a distinct condition listed under genitourinary diseases but cross-listed in depressive disorders.
- This was considered a breakthrough for women's health, but also invited controversy regarding the purported pathologisation of 'normal hormonal changes' and risks of over-diagnosis.
- Since the inclusion of PMDD in the DSM and ICD there has been a significant increase in interest, funding and research into the condition.

How can we diagnose PMDD?

- The DSM-5 diagnostic criteria for PMDD require the following to be fulfilled:
 - **Cyclicity of symptoms**
 - Cyclicity is key, with definite temporal relationship to cycle.
 - Must be present in the final week before the onset of menses, start to improve within a few days after the onset of menses and become minimal or absent in the week post menses.

- **Presence of at least 5 of 11 symptoms as listed here:**
- **Core symptoms (of which at least one must be present):**
 - Marked affective lability (e.g. mood swings, feeling suddenly sad or tearful, increased sensitivity to rejection).
 - Marked irritability or anger and increased interpersonal conflicts.
 - Markedly depressed mood, feelings of hopelessness, or self-deprecating thoughts.
 - Marked anxiety, tension and/or feelings of being keyed-up or on edge.
- **Additional symptoms**
 - Decreased interest in usual activities.
 - Poor concentration.
 - Lethargy, reduced stamina.
 - Marked change in appetite, overeating or food cravings.
 - Sleep changes – hypersomnia or insomnia.
 - A sense of being overwhelmed or out of control.
 - Physical symptoms such as breast tenderness, joint pains, bloating or weight gain.
- **Severity of impact**
 - Clinically significant impact must be present (i.e. causing distress and interfering with work, school or usual social activities or relationships with others).
- **Exclusion of PME**
 - Often the hardest part of diagnosis.
 - Should not be an exacerbation of an underlying psychiatric disorder such as anxiety, depression or personality disorder.
 - PMDD can co-occur with these conditions.
- **Confirmation with prospective daily ratings**
 - Diagnosis requires cycle diaries for at least two consecutive cycles.
 - If one of these cycles is atypical, complete a third.
- **Symptoms are not attributable to drugs / other medical condition**
 - Take a careful drug history and medical history to exclude influences of medication, drug abuse or a medical condition such as hyperthyroidism.

Suicidality in PMDD

- Women with PMDD must be considered high-risk for suicidality. Loss of impulse control and impaired interpersonal functioning are likely to contribute to vulnerability to suicide in women with PMDD.
- Studies suggest that cycle diaries, though crucial to accurate diagnosis, are often not completed. Women are often desperate for help by the time they speak to a healthcare professional and if there is extreme distress or suicidal ideation, delaying treatment by 2 months may be unacceptable.

Differential diagnoses, variances and other associations in PMDD

- Distinguishing PMDD from premenstrual exacerbation or other mental health or physical conditions can be difficult. The presence of a symptom-free window for at least a few days after menstruation is crucial.
- Cycle diaries and a symptom-free interval are essential to separate PMDD from the premenstrual exacerbation of another condition.
- Particular care should be taken to distinguish PMDD from emotionally unstable personality disorder and understanding how it may relate to trauma-related conditions.
- A history of exposure to trauma is more likely in women with PMDD and there can be overlap between the diagnostic criteria for these conditions. Evidence also suggests women with trauma exposure may experience worsening of trauma-related symptoms during periods of reproductive shift, e.g. postpartum and in the perimenopause. Understanding the interrelated

nature of lived experience and physical/psychological symptoms allows us to take a holistic, biopsychosocial approach to management of these issues.

- PMDD is common in the neurodivergent population. Consider if ‘missed’ neurodivergence may be present when diagnosing PMDD. Look for the presence of cyclical exacerbation or symptoms in neurodivergent patients.
- Although the DSM-5 criteria focus on the classical late luteal pattern of symptoms, it is becoming increasingly evident that the PMDs encompass women with other patterns of cyclical symptoms, e.g. exacerbation during ovulation, or symptoms starting a few days after menses.
- Pragmatic questions should be asked, particularly whether this patient is likely to benefit from cycle control to manage her pattern of physical and psychological symptoms. The focus should be on identifying patterns that repeat from month to month, and on shared decision-making regarding treatments that may reduce the severity and impact of symptoms.
- Given the high risk of suicidal ideation and attempts in this population, patients with PMDD in primary care can be referred to secondary care for diagnosis and MDT management of symptoms, if the practitioner is not confident in management.

8.1.6 Management of the premenstrual disorders

Lifestyle and diet

- All women with premenstrual disorders should be advised that lifestyle modification has been shown to improve symptoms.
- General measures to improve symptoms include:
 - Eating regular balanced meals every 2–3 hours that are rich in complex carbohydrates (theorised to increase central serotonin availability via increased tryptophan, with some trial data to support this).
 - Regular moderate aerobic exercise.
 - Regular sleep.
 - Stress reduction.
 - Smoking cessation and alcohol restriction, where applicable.
 - Cognitive behavioural therapy (CBT) should be routinely offered and can be useful alongside pharmacological approaches as well as being an alternative to medication.

Supplements and complementary medicine

- Vitamin B6 has been extensively studied and 100mg has shown weak superiority to placebo in meta-analysis. Higher doses may lead to neurological side-effects.
- Calcium supplementation at 600mg twice daily has shown some benefit in RCTs. There is some additional evidence supporting vitamin D supplementation.
- Magnesium is recommended by the International Association for Premenstrual Disorders (IAPMD) at 500mg daily following evidence of benefit.
- *Agnus castus* is the best researched supplement for PMS with encouraging evidence of benefit. There is no consistent dose recommendation, although the National Association for Premenstrual Syndrome (NAPS) flow chart suggests 20–40mg daily. There is no standardised quality-controlled preparation available.
- Acupuncture, St John’s wort, ginkgo, isoflavones and a number of other complementary and alternative medicines have a weak evidence base for efficacy but should currently not be recommended first-line.

Non-hormonal medications

- Menstrual cramping and joint pain can be managed with NSAIDs (unless contraindicated), e.g. ibuprofen, mefenamic acid or naproxen.
- SSRIs have a clear evidence base for efficacy and should be considered a first-line treatment.

- When used for PMDs, SSRIs seem to have a very rapid onset of action, with improvement seen within 24 hours and peaking at 48 hours. This suggests a different mode of action than when used in depression or anxiety.
- Starting doses are the same as for other indications and they appear equally effective when offered continually, or for the luteal phase only (i.e. given on days 15–28 of the cycle). Luteal phase dosing is off-licence.
- Adverse effects are present in around 50% of SSRI users, including sexual dysfunction, nausea, sleep disturbance and fatigue. Loss of libido can be particularly challenging for women with PMS/PMDD who may already be struggling in their relationships.
- Research is taking place into allopregnanolone modulators (blocking allopregnanolone at the GABA-A receptor). 5-alpha reductase inhibitors have also been studied, as they block the conversion of progesterone to allopregnanolone.
- Anecdotally, some women find antihistamines such as fexofenadine or H₂ antagonists such as famotidine helpful. There is currently no clear evidence to support their role in management. There are also potential overlaps between premenstrual disorders, hypermobility, mast-cell activation syndrome (MCAS) and autonomic nervous system dysfunction, e.g. POTS (postural orthostatic tachycardia syndrome), which may explain the efficacy of these medications in some patients.

Hormonal medications (including use of GnRH analogues)

- **Women with PMDs have usually experienced unwanted mood change with hormonal medications and are often nervous when a prescription is suggested.**
- It is important to reassure them that initial flare in symptoms is common with all options listed below but this usually settles within 6 weeks. **They should be reassured that they will be listened to if they wish to stop medication because they cannot tolerate it.**
- Patients may need to try several different options before finding one that suits them.
- Hormonal methods may be offered alongside, or as an alternative to, lifestyle and complementary therapies and SSRIs.
- Where hormonal medications fail to improve symptoms, patients should be referred to secondary care for consideration of GnRH analogues and/or surgery.

Combined oral contraceptives

- Ovarian suppression and modulation of hormonal fluctuations in the menstrual cycle are the main aims of hormonal therapies for PMDs.
- The combined oral contraceptive pill is an effective option when used in a tailored regimen. Evidence supports giving the COCP continuously (stopping only when breakthrough bleeding occurs) or bi-/tricycling (i.e. reducing the pill-free interval to every second or third pack).
- Breaks may need to be taken if persistent spotting occurs. When a break is taken, reducing this to 4 days rather than the typical 7 can minimise the risk of cycle-related symptoms.
- The best evidence base is for drospirenone-containing contraceptives, e.g. Yasmin / Lucette / Dretine / Yacella / Eloine.
- Evidence is increasing that Zoely, which contains estradiol and nomegestrol, is also well-tolerated in women with PMDs.

Estradiol

- **Inhibition of ovulation / cycle suppression can also be achieved with higher doses of transdermal oestrogen.**
- **This is particularly useful in women in whom the COCP is contraindicated, or in women at perimenopause who request HRT.**
- 100mcg patches of transdermal estradiol have been shown to be effective at suppressing ovulation and cycle-related symptoms.
- Contraception cannot be assumed.

- The endometrium requires protection. The following are all acceptable options to try – it is worth taking a detailed history of what previous synthetic and natural progestogens may have been tried and how they were tolerated. This may inform your choice of progestogen:
 - **A 52mg LNG-IUD** provides low-dose levonorgestrel. Around 10% of women with progesterone intolerance will not tolerate this device. Women should be advised that symptoms can flare initially but this generally settles within 6 weeks. They should be reassured the device can be removed if they remain intolerant, which should eliminate symptoms within 24 hours. Many women with history of mood change with contraception are fearful of a coil, as they cannot stop the treatment themselves – giving this clear reassurance often allows a woman to feel more comfortable in trying this method.
 - **Continuous Utrogestan** (micronised progesterone) is often better tolerated than synthetic progestins. 100mg nocte has historically been used – though the recent BMS Joint Guideline on Unscheduled Bleeding would now suggest that 200mg may be required to protect against endometrial hyperplasia with a dose of 100mcg of transdermal oestrogen.
 - **Sequential micronised progesterone** may be needed to ensure predictable bleeding in younger women. The NAPS and RCOG guidelines do not advise on dose, but usually this has been 200mg for 12 days. Again, recent BMS guidelines suggest 300mg may be required for adequate endometrial protection. Watch for flare of symptoms with progesterone initiation or withdrawal, which will make this pattern of prescribing unsuitable for many.
 - **Micronised progesterone** may be better tolerated vaginally as Cyclogest pessaries or 8% Crinone gel. Current evidence supports giving the same dose advised for the oral route. This route bypasses first-pass metabolism, avoiding conversion to allopregnanolone.
 - Some progestogen-intolerant women may struggle to take the higher doses of progesterone recommended by the most recent guidelines. **In these cases, consider referring to a specialist for further management.** The RCOG guidelines state: *“When using a short duration of progestogen therapy, or in cases where only low doses are tolerated, there should be a low threshold for investigating unscheduled bleeding.”*
 - **Slynd** (drospirenone 4mg) is also now available and though off-licence, has been included by the BMS in acceptable options for endometrial protection (based on the fact that drospirenone-containing COCPs contain 3mg as adequate progestogenic opposition for up to 30mg of ethinylestradiol). This option has not been incorporated into PMD guidelines so far but has shown promising results in progestogen-sensitive women.

Danazol

- Danazol is an androgenic steroid which has been shown to be effective in cycle suppression but is rarely advised for use due to its potential for irreversible masculinising side-effects.

GnRH analogues

- **When simpler measures have failed, patients can be referred to secondary care for consideration of GnRH analogues.** These are not generally initiated by the non-specialist, but once commenced are often covered by shared care arrangements and administered in primary care.
- GnRH analogues produce profound ovarian suppression and effectively induce medical menopause.
- They induce hypo-oestrogenic side-effects which require treatment.
- GnRH analogues are usually reserved for severe PMDD where surgical management is being considered. If symptoms do not respond within 12 weeks, the diagnosis of PMDD may need reviewing.
- Significant bone loss begins within 6 months of use. GnRH analogues are therefore only licensed for 6 months of continuous use.
- Add-back hormones must be prescribed if use is to be extended beyond this. They are usually started much earlier to prevent bone loss and avoid troublesome vasomotor symptoms.

- Continuous combined HRT (the oestrogen dose does not need to be 100mcg in this situation as the ovaries are already suppressed by the GnRH analogue), usually with 100mg micronised progesterone orally or vaginally, is generally prescribed. An alternative is tibolone 2.5mg which is often well-tolerated.
- RCOG guidelines advise DEXA monitoring at least annually if on long-term GnRH analogues.
- Women may notice an initial flare in symptoms as the LH and FSH generally rise before being downregulated and therefore suppressing ovulation.

Surgical treatment of PMDD

- **When treating severe PMS or PMDD and other methods have failed, or long-term GnRH therapy is required, hysterectomy and bilateral oophorectomy has been shown to be of benefit.**
- Surgery is not usually considered without preoperative use of GnRH analogues to test cure and ensure that HRT will be tolerated.
- Occasionally oestrogen alone will be administered during this period if tibolone or progesterone are not tolerated. The RCOG guidelines state that this should be on an individual basis due to concerns regarding endometrial hyperplasia. This is usually only done with specialist initiation whilst considering surgical treatment.

8.1.7 Useful resources for professionals and patients

- www.PMS.org.uk – the National Association for Premenstrual Syndromes includes a PDF guideline for patients and professionals, cycle diaries and useful resources for managing the condition.
- www.IAPMD.org – the International Association for Premenstrual Disorders has information for professionals and patients, including webinars and cycle diaries, and online peer support for people with PMDD around the world.
- www.rcog.org.uk – the RCOG Green-top Guidelines for managing premenstrual syndrome.

8.2 Perinatal mental health

8.2.1 Definition and risk factors

- The DSM-5 criteria specify that peripartum depression onset is during pregnancy or within 4 weeks after delivery.
- It is generally agreed, however, that onset can occur at any time within the 12 months following childbirth. This contrasts with the ‘baby blues’, which affect between 3 and 8 out of 10 women and are usually mild and transient.
- Risk factors for perinatal depression include:
 - prior depression or anxiety, including during a previous pregnancy
 - life stress
 - prior history of premenstrual disorders or adverse mood reaction with hormonal contraceptives
 - lack of social support
 - relationship difficulties, e.g. poor partner support
 - domestic violence
 - unintended pregnancy
 - history of trauma, e.g. childhood abuse
 - complications at birth, e.g. preterm delivery, infant health problems or need for intensive care
 - antenatal thyroid dysfunction or pregestational/gestational diabetes
 - longer time to conception
 - having two or more children
 - history of substance misuse
 - discontinuation of psychotropic medication prior to/during pregnancy.

- Where there is a personal history of past or present severe mental illness, or a family history of severe perinatal illness, there is a higher chance of postpartum psychosis in the first 2 weeks after childbirth.

8.2.2 Prevalence and impact

- During pregnancy, 12% of women will be affected by depression and 13% of women will be affected by anxiety. In the first year after birth, 15–20% will be affected.
- First-time mothers, adolescent mothers and those who have had a traumatic delivery may benefit from proactive support. Studies have shown that home health visits, telephone peer support and psychotherapy can help prevent postpartum depression.
- Evidence suggests that perinatal depression is often missed or undertreated in general practice.
- Severe depression in pregnancy is associated with increased rates of obstetric complications, sudden infant death syndrome, low birthweight and preterm delivery, self-harm and suicide attempts.
- Suicide remains a leading cause of maternal death in the first postpartum year.
- There is an association between depression in pregnancy and depression in the adolescent and young adult offspring.
- There may also be impairment in cognitive, behavioural and emotional development of a minority of infants born to mothers with perinatal depression.
- There may be wider impacts within the family, including effects on the woman's partner and other children.
- Many women feel shame about these conditions and may benefit from psychological support to process their experience.

8.2.3 Prognosis

- If depression is untreated during pregnancy, women have a seven-fold increased risk of postpartum depression when compared to women without antenatal depression.
- Postpartum depression often improves spontaneously after 2–3 months. One-third of women remain unwell 12 months after childbirth, and 13% at 2 years.

8.2.4 Diagnosis

- **Mental health should be assessed during the pregnancy booking appointment, and in all contacts through the antenatal and postnatal period.**
- The postnatal check should include screening questions, such as the ones below, to elicit whether the baby blues resolved within 10–14 days of birth, or whether low mood has persisted:
 - *“During the past month, have you often been bothered by feeling down, depressed or hopeless?”*
 - *“During the past month, have you often been bothered by having little interest or pleasure in doing things?”*
- If there is a positive response to the depression screening questions, or the mother is at risk of a mental health problem, consider further evaluation using the Patient Health Questionnaire (PHQ-9) or the Edinburgh Postnatal Depression Scale (EPDS).
- Women should be assessed for level of risk. If there are severe symptoms, or you believe mother or infant to be at risk, refer to a mental health professional.
 - Consider whether there is adequate social support, and ensure the woman and her partner or other support know where to seek further help if things deteriorate.
 - You may need to follow local safeguarding protocols if you have any concerns about risk of child harm or maltreatment.
- Consider differential diagnoses, e.g. bipolar disorder and OCD.
- Ask about the presence of confusion, delusions or hallucinations, which may indicate the onset of postpartum psychosis.

8.2.5 Managing perinatal depression

Women on antidepressants prior to pregnancy

- **Where pregnancy is planned, advise women that antidepressants may be used at any stage of pregnancy where clinically indicated, including when trying to conceive.**
- The risk of destabilising the woman's condition should be weighed up against the wish to reduce, change or stop antidepressants.
- Aim for the lowest effective dose, and a single drug is preferable to polypharmacy. Where prescribing is complex, seek specialist advice.
- Antidepressants should not be stopped abruptly.
- No antidepressant has been proven to cause birth defects.
 - SSRIs:
 - SSRIs have the largest body of safety data and can be considered a first-line choice. There is no evidence one SSRI is safer than any other. Studies suggest a small risk of fetal heart defects (3 in 100 vs. 2 in 100 in the background population).
 - SSRI use in pregnancy may lead to transient neonatal withdrawal causing central nervous system, motor, respiratory and gastrointestinal symptoms. Hospital delivery is usually advised.
 - Tricyclic antidepressants (TCAs):
 - Although usually considered a second-line treatment in pregnancy, they are safe for use if clinically indicated (for example, where a patient is stable on them and may be at risk of relapse if treatment is withdrawn or changed).
 - Data is more limited than for SSRIs.
 - Serotonin–noradrenaline reuptake inhibitors (SNRIs):
 - Data is even more limited than for SSRIs and TCAs.
 - Where it is clinically indicated, they can be considered safe to use or continue, particularly where a woman is stable on treatment and at risk of relapse if treatment is stopped or changed.
- Consider her plans for breastfeeding, as there are some risks associated with psychotropic medications in this instance.
- Detailed information on risks of individual antidepressants is available from the UK Teratology Information Service (www.uktis.org). Prescribing should be a shared care decision, including the patient.
- Consider referring women with severe current or previous mental health issues, who are planning pregnancy, to secondary mental health service for preconception counselling. Pregnancy and the puerperium are vulnerable periods for relapse or destabilisation.

Managing new-onset antenatal depression

- Low-risk women with new onset of depression during pregnancy may be managed in primary care following the recommendations on treatment detailed at the start of *Section 8.2.5*.
- Consider urgent referral to secondary mental health services:
 - If there is evidence of risk of harm to the woman or other people.
 - If there is evidence (or history) of bipolar disorder.
 - If there is a history of severe mental illness, including previous perinatal depression or puerperal psychosis in the woman or a first-degree relative.

Managing postnatal depression

- Refer for immediate assessment by secondary mental health services (within 4 hours) if a woman has sudden onset of symptoms suggestive of postpartum psychosis or is at immediate risk of harm to herself or her baby.
- Refer urgently to secondary mental health services:
 - If she is severely depressed.

- If she shows signs of self-neglect or being unable to look after her baby.
- If there is a possible diagnosis of bipolar disorder.
- If there is a history of severe mental illness, including perinatally.
- Where a woman is safe to be managed in primary care, take into account her preferences when prescribing.
- Information is available from the UK Drugs in Lactation Advisory Service (UKDILAS) on 0330 770 8564.
- An SSRI, TCA or SNRI can safely be used in the postnatal period.
- No psychotropic medication has a licence for use in breastfeeding mothers, so informed consent should be sought and documented.
- Paroxetine and sertraline are usually safer choices in breastfeeding mothers. The lowest effective dose should be used, and babies should be monitored for drowsiness, poor feeding and behavioural changes.

8.2.6 Postpartum psychosis

- Postpartum psychosis is rare, affecting 1–2 in 1000 women.
- It usually presents suddenly, within 2 weeks of delivery.
- Symptoms include severe mood swings, delusions, confusion and hallucinations. Distorted thoughts and behaviours may involve the baby and place it at risk.
- Recurrence in subsequent deliveries is common.

8.2.7 Bipolar postpartum depression

- Bipolar disorder may present postnatally, or an existing diagnosis may relapse in this period.
- 21.4–54% of women with postpartum depression have a diagnosis of bipolar disorder.
- These women are often younger in age, with earlier onset of symptoms after birth and some atypical depressive features.
- There may be a history of bipolar disease in first-degree relatives.
- It is important to identify bipolar symptoms, as treatment with antidepressants may trigger manic symptoms.
- These patients are usually managed by specialist mental health teams. Treatment often includes use of mood stabilisers, e.g. lithium, quetiapine and lamotrigine.

8.2.8 Perinatal obsessive–compulsive disorder

- Prevalence of obsessive–compulsive disorder (OCD) is higher in the perinatal population than in the general population.
- Clinical features in the perinatal period are likely to include concerns about harm to the infant, with contamination and cleaning/checking compulsions particularly common.
- Research suggests CBT with exposure and response prevention is particularly effective.
- There is also limited evidence for efficacy of SSRIs.
- Patients should be referred to specialist mental health services for assessment and treatment.

8.3 Hormonal contraception and mood disorders

- **Hormone-containing medications can often induce mood change in susceptible women.** Where cyclical, this falls under ‘Variant PMDs – progestogen-induced’ as detailed in *Section 8.1.4*.
- Where continuous administration of hormonal medication leads to mood change, this is usually considered an adverse effect rather than a form of premenstrual disorder. Colloquially, we often refer to this as progestogen intolerance or sensitivity.

- Management follows the NAPS and RCOG guidelines for PMS and PMDD. Many women will find they tolerate drospirenone- or norgestrel-containing COCPs better or will improve when offered high-dose (100mcg) transdermal oestrogen alongside an LNG-IUD coil or anovulatory progestogen such as Slynd (drospirenone). Desogestrel is less often tolerated by progestogen-sensitive women, but may also be given. It can be used off-licence, in a double dose of 150mg, alongside transdermal oestrogen to provide both contraception and endometrial protection (though off-licence, this option is included in the BMS Joint Guidelines on Unscheduled Bleeding).
- Micronised progesterone is not contraceptive so is unsuitable for use for this indication.

8.4 Neurodiversity in women and girls

8.4.1 What is neurodiversity and who is affected?

- Neurodivergence is a term that describes the natural variation in human brain functioning and cognitive processing.
- Autism prevalence is estimated at 1–2% but is growing, likely due to improved recognition – especially amongst those with normal range IQ, and due to broadening of diagnostic criteria over time.
- Variations from the majority or ‘neurotypical’ population include:
 - autism spectrum disorder (ASD)
 - attention deficit hyperactivity disorder (ADHD)
 - dyslexia
 - dyspraxia (developmental coordination disorder)
 - Tourette syndrome
 - sensory processing differences.
- Diagnosis of neurodivergence is often missed or delayed in women and those assigned female at birth. Common symptom profiles were focused on male presentation, and biological and social factors can mean that the conditions present differently in females.
- A predictive model based on population data has suggested that 39% more women should be diagnosed with ASD than currently are.
- Diagnosis in adulthood is often more challenging, as there may be a lack of developmental history and individuals have learnt strategies to camouflage (mask) difficulties.
- Neurodivergent women and girls often mask more successfully than their male counterparts, but may struggle to continue to do so at reproductive milestones such as menopause.
- The considerable cognitive effort required to mask difficulties relating to neurodivergence leads to increased rates of stress, anxiety and depression.
- Individuals with ‘milder’ presentation may be more prone to late or missed diagnosis and have developed more coping strategies which ‘hide’ the diagnosis. For example, women with ADHD often become prone to work-related burnout, as challenges with executive function such as planning, prioritising, scheduling and multitasking can lead to overload and overwhelm. This may present clinically as anxiety, insomnia or depression, but deeper questioning may reveal relevant detail pointing to the need to consider neurodivergence.
- Consideration of the wider clinical picture can help in identifying possible ‘missed’ neurodivergence.
- Neurodivergent women and girls are more likely to also have:
 - Eating disorders (multiple studies reveal an overrepresentation of autism or autistic traits in the eating disorder population).
 - Premenstrual disorders such as PMS or PMDD (some research suggests that autistic individuals may be 2–3 times more likely to report cyclical mood change consistent with PMDD).

- Heightened sensory sensitivity, emotional dysregulation and executive function challenges at certain points in their menstrual cycle or at reproductive milestones such as menopause.
- Family members with existing ASD or ADHD diagnoses (heritability is estimated between 64% and 91%).
- Neurodivergence is not a mental health condition, but neurodivergent individuals are at a high risk of developing mental health problems, with depression and anxiety problems predominating, and suicide rates substantially increased.

8.5 Eating disorders and body dysmorphic disorder

8.5.1 Introduction

- Although eating disorders can present at any age, risk is highest between 13 and 17 years of age.
- It is estimated that over 725 000 people in the UK have an eating disorder. This is based on hospital admissions, so is likely to be a significant underestimate.
- The types of eating disorders as defined in the DSM-5 include the following:

Anorexia nervosa

- The lifetime prevalence of anorexia nervosa (AN) in females is between 2 and 4%.
- AN has a higher mortality rate than any other mental health disorder. 20% of deaths are due to suicide. The crude mortality rate is 5.1 deaths per 1000 person years.
- Clinical features of AN include:
 - Restricted energy intake or persistent behaviour which prevents weight gain and leads to a significantly low body weight.
 - Body image is disturbed, with denial of the seriousness of the current low body weight, or undue influence of body weight or shape on self-evaluation.
 - There is an intense fear of gaining weight, despite being underweight.
 - BMI specifiers were added in 2013:
 - Mild: BMI 17–18.5kg/m²
 - Moderate: BMI 16–16.9kg/m²
 - Severe: BMI 15–15.9kg/m²
 - Extreme: BMI <15kg/m²
 - The available evidence has questioned the reliability and clinical validity of these definitions.
- Although females with AN may present with hormonal disturbance and amenorrhoea, it is no longer included in the diagnostic criteria. However, periods and growth/puberty are often disturbed. Where amenorrhoea persists beyond 6 months there is risk of reduced bone density.
- Physical signs may include dry skin and hair loss, bradycardia, orthostatic hypotension, hypothermia, loss of muscle strength, constipation, fainting and fatigue.

Bulimia nervosa

- Recurrent (at least once weekly for 3 months) episodes of uncontrolled eating of an abnormally large amount of food.
- Binges are followed by inappropriate compensatory behaviours (induced vomiting, diet pill or laxative abuse) or excessive exercise.
- Self-evaluation is unduly influenced by body shape/weight, and often there is an intense fear of gaining weight.
- There may be persistent preoccupation with and cravings for food, then guilt and shame about bingeing and purging.

- Bulimia may go undiagnosed for years, as individuals may maintain a normal body weight and appear to eat normally in social situations.
- Physical signs may include knuckle calluses from recurrent induced vomiting (Russell’s sign), salivary gland enlargement and dental enamel erosion.

Binge eating disorder

- Recurrent episodes of binge eating without any compensatory behaviours.
- Episodes associated with distress, guilt and marked loss of control.
- Individuals are often overweight or obese.

Avoidant/restrictive food intake disorder (ARFID)

- This is also referenced as ‘selective eating disorder’ in the DSM.
- Avoidance of food or restrictive pattern of eating based on certain food characteristics or aversive consequences.
- Leads to significant weight loss, nutritional deficiency, or dependence on supplements.

Pica

- Persistent eating of non-nutritive, non-food substances (for at least 1 month).
- Inappropriate to the developmental level.

Rumination

- Repeated regurgitation of food which may be re-chewed, re-swallowed, or spat out.
- Not due to a medical condition or better explained by another disorder.

Other specified feeding or eating disorder (OSFED)

- This is where there is clinically significant distress, but the full criteria for feeding and eating disorders are not met. Includes the following:
 - Atypical AN refers to restrictive disordered eating in people not at an extremely low body weight
 - Subthreshold bulimia or binge eating disorder
 - Purging disorder
 - Night eating syndrome.

Unspecified feeding or eating disorder (UFED)

- This covers disordered eating not more accurately captured by OSFED.

8.5.2 How can we diagnose eating disorders?

- **Eating disorders can be difficult to diagnose, especially in primary care.**
- Patients are often slow to present and may hide or fail to disclose symptoms. They may not think they have an eating disorder.
- The SCOFF questionnaire is a short, focused screening tool to identify anorexia nervosa or bulimia nervosa:
 - “Do you ever make yourself feel **S**ick because you feel uncomfortably full?”
 - “Do you worry that you have lost **C**ontrol over how much you eat?”
 - “Have you recently lost more than **O**ne stone in a 3-month period?”
 - “Do you believe yourself to be **F**at when others say you are too thin?”
 - “Would you say that **F**ood dominates your life?”
- Severe malnutrition and purging behaviours can cause cardiovascular instability or severe electrolyte disturbance – have a low index for assessing clinical signs. Emergency admission may be required; for example, where there is syncope, pre-syncope or severe abdominal pain. Check for risk of self-harm and suicide.

- Examination should ideally include calculation of BMI, temperature (hypothermia), BP (including lying/standing), hydration status, peripheral circulation. Look for muscle wasting (consider the Sit up-Squat-Stand (SUSS) test).
- Consider checking FBC, erythrocyte sedimentation rate (ESR), U&Es, LFT, blood glucose, creatinine and urinalysis and ECG if there is significant malnutrition or purgative behaviour. Calcium, magnesium, phosphate, thyroid function test (TFT), B12, folate and ferritin are sometimes also requested.
- Consider risk of insulin misuse in diabetic patients: often missing or reducing doses to induce weight loss. A high HbA1c, or history of recurrent diabetic ketoacidosis may raise suspicion of this.
- Differential diagnoses include inflammatory bowel disease, coeliac disease, mood disorders, drug misuse, thyroid disorders and malignancy.

How can we manage eating disorders?

- **Assess the need for emergency admission if there is serious medical or psychiatric risk.**
- Refer immediately to an eating disorder service for specialist assessment and management. Shared care agreements may be in place, particularly in chronic eating disorders.
- Whilst awaiting specialist assessment arrange regular review as appropriate.
- Have a low threshold for concern – people with eating disorders can appear deceptively well despite being medically unwell.
- Consider emergency admission if:
 - BMI or body weight rapidly falling (e.g. >1kg per week)
 - cardiovascular instability, e.g. bradycardia <40 bpm, tachycardia on standing, prolonged QT on ECG, or hypotension
 - hypothermia
 - reduced muscle power
 - concurrent infection
 - rapid deterioration
 - abnormal bloods
 - acute mental health risk, e.g. suicide attempt or serious self-harm.
- Compulsory admission may be required – seek specialist advice if you have serious concerns about a patient's safety but they do not consent to admission.
- The Royal College of Psychiatrists has released guidelines on assessing the impending risk to life in feeding and eating disorders. This document provides a red/amber/green rating to help us assess when urgent help may be needed. This is adapted from the previous MARSIPAN and JUNIOR MARSIPAN framework. The framework combines assessment of clinical risk factors with consideration of patient motivation, engagement with healthcare, and the presence of support around them.
- It is important to note that it is not only patients with AN who may present with immediate risk to life – for example, bulimic patients risk life-threatening electrolyte disturbances and gastrointestinal complications.

Bone health in eating disorders

- **Women with AN are 150–300% more likely to have fractures due to significantly lower bone mineral density than healthy control women.**
- Weight restoration is of key importance in the underweight to protect bone health.
- The long-term effects of bisphosphonates in this population are still unknown. The decision to start hormonal treatment for low bone density in a patient with an eating disorder should be taken with specialist advice.

Body image in women

- Body image is a frequent concern for women, with evidence that pregnancy, cancer and gynaecological conditions such as PCOS and reproductive milestones such as menopause are correlated with increasing body image concerns.
- Clinicians should be sensitive to the wider psychosocial pressures on women and consider referral for psychological support where women experience distress related to body image.

Body dysmorphic disorder in women

- Body dysmorphic disorder (BDD) is common but under-recognised. It affects women more than men. There is up to 49% heritability.
- BDD and OCD share genetic vulnerability. BDD may co-exist with OCD. There is also association with substance misuse and social anxiety disorder.
- Patients may not spontaneously disclose their concerns for fear of shame and negative judgement. Clinicians therefore may need to ask direct questions to identify the condition.
- It is defined in the DSM-5 as a preoccupation with perceived defects in one's physical appearance that to other people appear non-existent or only slight.
- The appearance preoccupation can trigger excessive repetitive behaviours (such as mirror checking, excess grooming, skin picking) or repetitive mental acts. To be diagnosed, the behaviours must cause impairment or distress.
- It is associated with marked functional impairment, poor quality of life and high rates of suicidality.
- Most women with BDD will seek cosmetic surgery treatment for their BDD concerns, but such treatment virtually never improves BDD symptoms and often makes them worse.
- Patients with suspected BDD should be referred to specialist mental health services for diagnosis and management. Treatment relies on psychotherapeutic approaches and pharmacotherapy, e.g. SSRIs.

8.6 Further reading

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