

ANSWER KEY WITH EXPLANATIONS FOR CHAPTER 23

Skill Drill Answers

SD 23.1: Hormone-Effect Matching

1. **A** — Estrogen causes endometrial proliferation and bone protection
 2. **B** — Progesterone causes secretory transformation and maintains pregnancy
 3. **C** — Testosterone causes male characteristics and muscle mass
 4. **D** — Aldosterone causes sodium retention and potassium excretion
 5. **E** — Cortisol has anti-inflammatory and metabolic effects
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SD 23.2: True/False with Correction

1. **T** — Unopposed estrogen stimulates endometrial proliferation, increasing cancer risk.
 2. **F** — Tamoxifen is an estrogen **ANTAGONIST** in breast tissue (that's why it treats breast cancer).
 3. **F** — GnRH agonists cause initial testosterone **SURGE** (flare) before suppression from receptor downregulation.
 4. **F** — Chronic glucocorticoids must be **TAPERED** to avoid adrenal crisis.
 5. **T** — Spironolactone blocks androgen receptors in addition to mineralocorticoid receptors.
 6. **F** — Transdermal estrogen has **LOWER** thrombotic risk (bypasses first-pass hepatic effects on clotting factors).
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SD 23.3: Fill in the Blank

1. **LH**
2. **endometrial**
3. **dihydrotestosterone (DHT)**

4. **HPA (hypothalamic-pituitary-adrenal)**
 5. **hypothalamus**
 6. **mineralocorticoid**
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SD 23.4: Drug Class Matching

1. **A** — Ethinyl estradiol + levonorgestrel is a combined hormonal contraceptive
 2. **B** — DMPA is a progestin-only contraceptive
 3. **C** — Tamoxifen is a SERM
 4. **D** — Leuprolide is a GnRH agonist
 5. **E** — Finasteride is a 5α -reductase inhibitor
 6. **F** — Prednisone is a glucocorticoid
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SD 23.5: SERM Tissue Effect Matching

Tamoxifen:

1. **X** — Antagonist in breast tissue
2. **A** — Agonist in bone
3. **A** — Agonist in endometrium

Raloxifene: 4. **X** — Antagonist in breast tissue 5. **A** — Agonist in bone 6. **X** — Antagonist in endometrium

SD 23.6: Rapid Recall

1. **Clomiphene**
 2. **Dexamethasone**
 3. **Ulipristal (Ella)**
 4. **Spironolactone**
 5. **Osteoporosis**
 6. **Raloxifene**
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SD 23.7: Complete the Sentence

1. **contraindications**
 2. **surge**
 3. **tapered**
 4. **50%**
 5. **anti-androgenic**
 6. **does not increase**
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SD 23.8: Contraceptive Method Matching

1. **C** — Copper IUD is most effective emergency contraception
 2. **D** — DMPA is injection every 3 months
 3. **E** — Etonogestrel implant is subdermal rod for 3 years
 4. **B** — Levonorgestrel IUD has local progestin effect
 5. **A** — Combined OCP contains estrogen and progestin daily
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SD 23.9: Anti-Androgen Mechanism Matching

1. **A** — Leuprolide is a GnRH agonist
 2. **C** — Enzalutamide is an androgen receptor antagonist
 3. **D** — Finasteride is a 5α -reductase inhibitor
 4. **B** — Degarelix is a GnRH antagonist
 5. **E** — Abiraterone is a CYP17 inhibitor
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SD 23.10: Glucocorticoid Comparison

- 1** — Hydrocortisone (lowest potency)
 - 2** — Prednisone
 - 3** — Methylprednisolone
 - 4** — Dexamethasone (highest potency)
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SD 23.11: Adverse Effect Identification

1. **T** — Tamoxifen increases thromboembolism risk
 2. **F** — Glucocorticoids cause HYPERGLYCEMIA, not hypoglycemia
 3. **T** — Spironolactone's anti-androgenic effects cause gynecomastia
 4. **T** — Anabolic steroids suppress the HPG axis, reducing endogenous testosterone
 5. **F** — Estrogen-containing contraceptives are CONTRAINDICATED in migraine with aura (stroke risk)
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SD 23.12: Clinical Scenario Drug Selection

1. **C** — Raloxifene treats osteoporosis without increasing breast cancer risk (unlike estrogen)
 2. **C** — Levonorgestrel IUD (progestin-only) is safe; estrogen-containing methods contraindicated in smokers >35
 3. **B** — Leuprolide (GnRH agonist) for androgen deprivation in prostate cancer
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Case Study Answers

Case 23.1: Hormone Replacement Therapy

Question 1. Progestin must be included because she has an intact uterus. Unopposed estrogen stimulates endometrial proliferation, significantly increasing the risk of endometrial hyperplasia and cancer. Progestin opposes this effect by inducing secretory transformation and preventing proliferation. Women who have had a hysterectomy can safely take estrogen alone.

Question 2. Expected benefits include relief of vasomotor symptoms (hot flashes, night sweats), improvement in vaginal atrophy and dryness (reducing dyspareunia), improved sleep, prevention of bone loss and reduction in fracture risk, and possible improvement in mood and quality of life. HRT is the most effective treatment for menopausal symptoms.

Question 3. Risks to discuss include increased venous thromboembolism (DVT, PE), small increased risk of breast cancer with long-term combined HRT (>5 years), small increased risk of stroke, and gallbladder disease. The risks are generally low in healthy women within 10 years of menopause and under age 60. The decision should be individualized based on symptom severity and risk factors.

Question 4. Transdermal estrogen (patch or gel) bypasses first-pass hepatic metabolism, reducing the effect on clotting factor synthesis. Studies suggest lower thrombotic risk compared to oral estrogen. This route is preferred for women with elevated thromboembolism risk but without absolute contraindication to estrogen.

Case 23.2: Chronic Glucocorticoid Therapy

Question 1. This patient is experiencing multiple glucocorticoid adverse effects: cushingoid appearance (moon facies, central obesity from fat redistribution), significant weight gain (15 kg), and steroid-induced diabetes/hyperglycemia (fasting glucose 180 mg/dL). He likely also has HPA axis suppression, and other complications such as osteoporosis, hypertension, and thin skin may be present but not mentioned.

Question 2. After 8 months of prednisone 20 mg daily, his HPA axis is suppressed. The adrenal glands have atrophied from lack of ACTH stimulation and cannot produce adequate cortisol. Abrupt discontinuation would cause adrenal insufficiency (Addisonian crisis)—hypotension, fatigue, nausea, potentially cardiovascular collapse. Gradual tapering allows the HPA axis to recover.

Question 3. Preventive measures should have included calcium and vitamin D supplementation for bone protection, bisphosphonate therapy if high fracture risk, blood glucose monitoring with early intervention for hyperglycemia, blood pressure monitoring, PPI or H2 blocker for GI protection (especially if concurrent NSAID use), Pneumocystis prophylaxis if high-dose therapy (≥ 20 mg prednisone equivalent for ≥ 1 month), and steroid-sparing agents to minimize glucocorticoid dose.

Question 4. Patients on chronic glucocorticoids have suppressed HPA axes and cannot mount an adequate cortisol response to surgical stress. He requires stress-dose steroids—typically hydrocortisone 50-100 mg IV before surgery, then tapered over 1-2 days back to his baseline dose. Without this, he could develop intraoperative hypotension or postoperative adrenal crisis.

Diagram/Table Exercise Answers

DT 23.1: SERM Tissue Selectivity

1. Tamoxifen acts as an estrogen **antagonist** in breast tissue.
 2. Tamoxifen acts as an estrogen **agonist** in bone, helping maintain density.
 3. Tamoxifen's agonist effect in the endometrium increases risk of **endometrial cancer (hyperplasia)**.
 4. Raloxifene differs from tamoxifen by having **antagonist** activity in the endometrium.
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DT 23.2: Androgen Deprivation Therapy

1. GnRH agonists (like **leuprolide**) cause testosterone suppression through receptor downregulation.
2. Enzalutamide blocks the **androgen** receptor.
3. Finasteride blocks the enzyme **5 α -reductase**, preventing DHT formation.
4. The initial testosterone surge with GnRH agonists is called "**flare**."

DT 23.3: Glucocorticoid Comparison Table

Drug	Relative Glucocorticoid Potency	Relative Mineralocorticoid Potency	Duration	Common Use
Hydrocortisone	1	1	Short	Adrenal insufficiency replacement
Prednisone	4	0.8	Intermediate	Anti-inflammatory
Methylprednisolone	5	0.5	Intermediate	IV pulse therapy
Dexamethasone	25	0	Long	Cerebral edema, suppression testing

DT 23.4: Contraceptive Comparison Table

Method	Hormones	Mechanism	Key Consideration
Combined OCP	Estrogen + progestin	Suppresses ovulation	Estrogen contraindications (DVT, migraine w/aura)
Progestin-only pill	Progestin only	Thickens cervical mucus	Safe with estrogen contraindications
Levonorgestrel IUD	Local progestin	Thickens mucus, endometrial atrophy	Long-acting (5-7 years), reversible
Copper IUD	None	Spermicidal, prevents fertilization	Most effective EC, no hormones
DMPA injection	Progestin	Suppresses ovulation	Delayed fertility return

Multiple-Choice Question Answers

Question 1. Answer: B

Women with an intact uterus require progestin with estrogen to prevent endometrial hyperplasia and cancer. Unopposed estrogen is only safe in women who have had a hysterectomy.

Question 2. Answer: B

Tamoxifen is an estrogen antagonist in breast tissue, blocking estrogen receptor signaling that drives ER-positive breast cancer growth. It has agonist effects in other tissues (bone, endometrium).

Question 3. Answer: B

Combined oral contraceptives work primarily by suppressing the LH surge, thereby preventing ovulation. Secondary mechanisms include cervical mucus thickening and endometrial changes.

Question 4. Answer: D

Dexamethasone has approximately 25× the glucocorticoid potency of hydrocortisone, making it the most potent. The order is: hydrocortisone (1) < prednisone (4) < methylprednisolone (5) < dexamethasone (25).

Question 5. Answer: C

Finasteride inhibits 5α-reductase, blocking conversion of testosterone to the more potent DHT. This shrinks prostate tissue in BPH and slows male pattern baldness.

Question 6. Answer: D

Estrogen-containing contraceptives (COC, patch, ring) are contraindicated with history of VTE. The levonorgestrel IUD is progestin-only with minimal systemic absorption, making it safe in this patient.

Question 7. Answer: B

Raloxifene is an estrogen antagonist in both breast AND endometrium, unlike tamoxifen which has agonist activity in endometrium. This makes raloxifene safer regarding endometrial cancer risk.

Question 8. Answer: B

The initial testosterone surge from GnRH agonists can cause tumor flare in prostate cancer. Concurrent anti-androgen therapy (bicalutamide) blocks testosterone effects during this period. After several weeks, testosterone is suppressed and the anti-androgen may be discontinued.

Question 9. Answer: B

Cushingoid appearance (moon facies, central obesity, buffalo hump) results from glucocorticoid excess, whether endogenous (Cushing's syndrome) or exogenous (iatrogenic from prednisone therapy).

Question 10. Answer: B

Clomiphene blocks estrogen receptors in the hypothalamus, eliminating negative feedback. This stimulates GnRH release, increasing LH and FSH, which stimulates ovarian follicle development and ovulation.

Question 11. Answer: B

Spironolactone has anti-androgenic effects by blocking androgen receptors (and to a lesser extent inhibiting testosterone synthesis). This makes it useful for hirsutism and acne in women.

Question 12. Answer: C

Finasteride reduces PSA by approximately 50%. To estimate the "true" PSA in a patient on finasteride, double the measured value. $2.0 \times 2 = 4.0$ ng/mL.

Question 13. Answer: C

Chronic glucocorticoid therapy suppresses the HPA axis. Abrupt discontinuation can cause adrenal crisis. Gradual tapering allows recovery of endogenous cortisol production.

Question 14. Answer: B

Dexamethasone has negligible mineralocorticoid activity (sodium/water retention), making it preferred when fluid retention is undesirable, such as in cerebral edema where brain swelling must be minimized.

Question 15. Answer: C

Ulipristal is a progesterone receptor modulator that primarily works by delaying or inhibiting ovulation. It is not an abortifacient and does not prevent implantation of an established pregnancy.

Question 16. Answer: C

Castration-resistant prostate cancer has progressed despite low testosterone from GnRH agonist therapy. Second-generation antiandrogens like enzalutamide have higher affinity for the androgen receptor and are effective in this setting.