Radiobiology practise questions 2025

Concepts

1. What are the 5 "R"s of radiobiology?
2. Which of the 4 "R"s describe processes that take place during fractionated radiotherapy?
3. Explain the difference between "direct" and "indirect" DNA damage from radiation
4. Do X-rays cause most indirect or direct DNA damage?
5. Do carbon ions cause most indirect or direct or DNA damage?
6. Approximately how many DNA double strand breaks per cell are caused by 1Gy X-rays?
7. What makes radiation-induced DNA double strand breaks more toxic than spontaneously arising double strand breaks?
8. Why is radiation effective in killing cells despite the relatively small amount of energy deposited?
9. List 3 kinds of cell death that can be observed after radiation
10. Which kind of cell death is most commonly responsible for loss of reproductive capability after radiation?
11. Which laboratory assay is best to use to measure radiosensitivity of a cell line growing as a monolayer?
12. Give an example of an "ex vivo" and "in vivo" clonogenic assay.
13. Which parameter is best used to describe radiosensitivity of cells?
14. What are the two main processes in cells that repair DNA double strand breaks?
15. If DNA-PK is recruited to a DNA double strand break, which repair process is typically started?
16. If ATM is recruited to a DNA double strand break, which repair process is typically started?
17. Which of the two main DNA double strand break repair processes are most likely to introduce errors?
18. Which of the two main DNA double strand break repair processes always repairs most of the breaks?
19. Which of the two main DNA double strand break repair processes causes severe radiosensitivity if you disrupt it?
20. Explain why cells typically become a little more resistant to radiation as they traverse S-phase.
21. Name 2 other processes than DNA repair that are activated by ATM after radiation.
22. Draw a typical cell survival curve, indicating
    i. Scale and unit of x- and y-axis
    ii. SF2
    iii. α/β

23. Which mathematical model adequately represents cell survival data at doses 1-6Gy, has only 2 parameters to fit, and provides a framework commonly applied to model fractionation effects in the clinic? Provide name and equation.
24. Which parameter is used to describe the bendiness of the cell survival curve?
25. How does the shape of a curve with high α/β value compare to one with a low α/β value?
26. Does a high or a low α/β value indicate high capacity to repair damage between fractions of radiation?
27. Explain "the principle of equal effect per fraction".
28. What would your survival curve look like if you instead of one large dose of radiation gave multiple doses of 2Gy?
29. Add a survival curve in your diagram that might represent the same cells irradiated in the absence of oxygen.
30. What is the "oxygen enhancement ratio" (OER)?
31. Use the diagram in #28 to indicate how you would quantify OER.
32. Explain why oxygen is a powerful radiosensitizer.
33. Explain why the presence of oxygen is less important for higher LET radiation.
34. What is a "Bragg peak"?
35. Why do particles typically offer better opportunity than photons for delivering precise dose distribution in patients?
36. Define "Relative Biological Effectiveness" (RBE).
37. Define "Linear Energy Transfer" (LET).
38. How does RBE vary with increasing LET?
39. Explain why RBE reaches a maximum at 100keV/μm
40. Give two examples of radiation types commonly referred to as "low LET".
41. Give two examples of radiation types commonly referred to as "high LET".
42. For irradiated "early responding" normal tissues, how does radiation dose affect:
    i. Severity of the reaction
    ii. Time of peak reaction
    iii. When the reaction resolves
43. Provide 2 examples of "early responding" normal tissues
44. Provide 2 examples of "late responding" normal tissues
45. For irradiated "late responding" normal tissues, how does radiation dose affect severity of the reaction?
46. Describe how reactions in "late responding" normal tissues most typically change over time.
47. What inherent property of an irradiated "acute responding" normal tissue determines the time it takes to first observe a reaction?
48. How does lowering dose-rate affect the shape of the cell survival curve?
49. Tissue A is characterized by low α/β value, tissue B by high α/β value. Which one will benefit (i.e. be spared) most by lowering the dose-rate?

50. Define “Hyper”-, “Hypo”- and “Accelerated” fractionation schedules
51. Is SBRT an example of extreme hyper-, hypo- and/or accelerated fractionation strategy?
52. Tissue A is characterized by low α/β value, tissue B by high α/β value. Which one will benefit (i.e. be spared) most by lowering the dose per fraction?
53. An organ at risk (OAR) is characterized by low α/β value, the tumor by high α/β value. Would the therapeutic ratio increase or decrease with hypofractionation?
54. Which radiobiological “R” does accelerated fractionation schedules combat?
55. Does acceleration typically exacerbate acute responding tissue reactions?
56. Does acceleration typically exacerbate late responding tissue reactions?
57. Draw a typical dose-response curve that shows probability of tumor control as a function of dose.
58. Indicate the γ_{50} value in your diagram.
59. What does the γ_{50} represent?
60. Add a typical curve illustrating probability of normal tissue complications.
61. In which direction would adding a general radiosensitizer move the curves?
62. In which direction would adding a general radioprotector move the curves?
63. How would adding a tumor-specific radiosensitizer move the curves?
64. How would adding a normal tissue-specific radioprotector move the curves?
65. What is the “therapeutic ratio”?
66. Define “driver” and “passenger” genetic alteration in cancer.
67. Describe how targeting EGFR in combination with radiotherapy
    i. Targets a hallmark of cancer
    ii. Represents targeting “Oncogene Addiction”
    iii. Combats a radiobiological “R”
68. Name 2 cell types damaged by radiation that are thought to be important for mediating “late responding” tissue reactions.
69. Several studies report the risk of second cancer 5 years after radiotherapy, and compare this risk to patients not treated with radiotherapy. Is this a robust measure of radiation-induced cancers? Why / why not?
70. For most organs, how does the risk of developing a radiation-induced cancer depend on the radiation dose?
71. Giving high-dose rate brachy therapy for prostate cancer in 1 fraction was shown to result in loss of tumor control compared to 2 or more fractions. Which radiobiological “R”(s) might have contributed to the inferior result and why?
72. Might it be beneficial to administer a vasodilating drug before, during or after radiotherapy? Explain why.
73. Might it be beneficial to administer a vasodisrupting drug before, during or after radiotherapy? Explain why.
74. Which (rarely used) in vivo assay is considered the gold standard for comparing the potential for achieving cure of different treatments?

75. Describe the (theoretical) concept of "parallel" and "serial" organisation of different normal tissues. Give an example of an organ that would typically fit in each category.
76. Name 3 different experimental strategies (i.e. principles) to combat tumor hypoxia in patients.
77. What is the difference between a prognostic and a predictive factor?
78. Name 4 principles by which chemotherapy or a molecular targeted agent can increase the therapeutic ratio
79. Give an example of how chemotherapy can improve outcomes through spatial cooperation with radiotherapy
80. Explain how chemotherapy could enhance the efficacy of radiotherapy through modulating a radiobiological "R".

Calculations

1. A treatment is given as 70Gy in 35 fractions (Mon-Fri) to a head and neck cancer (α/β = 10) and surrounding connective tissue (α/β for fibrosis = 2).
    a. What is the EQD2 for the tumor?
    b. What is the EQD2 for the connective tissue?
2. A treatment is given as 70.2Gy in 39 fractions to a head and neck cancer (α/β = 10) and surrounding connective tissue (α/β for fibrosis = 2).
    a. What is the EQD2 for the tumor?
    b. What is the EQD2 for the connective tissue?
3. A treatment is given as 20 fractions of 3Gy to a prostate cancer (α/β = 1.5) and rectum (α/β for ulcer = 5).
    a. What is the EQD2 for the tumor?
    b. What is the EQD2 for the rectum?
4. A treatment is given as 10 fractions of 3Gy to a prostate cancer (α/β = 1.5) and rectum (α/β for ulcer = 5).
    a. What is the EQD2 for the tumor?
    b. What is the EQD2 for the rectum?
5. A treatment like in #1 was planned. Due to an error, 4 Gy per fraction was delivered the first week (5 days).
    a. If you continue the rest of the treatment with 2Gy per fraction, what will be the EQD2 for the tumor?
    b. If you continue the rest of the treatment with 2Gy per fraction, what will be the EQD2 for the connective tissue?
    c. Your colleague suggests continuing the treatment with 2Gy fractions until you reach the same effect on the tumor as originally planned. How many more fractions will you give?
    d. What is the EQD2 for this treatment on the normal tissue?

e. You figure it is better to keep the probability of toxicity the same. How many 2Gy fractions will you give then?
f. What is the % reduction in EQD2 to the tumor with this schedule?
g. How much will the probability of tumor control drop with this schedule?
h. You decide that the best solution is probably to correct your hypofractionation error with hyperfractionation. To accommodate this, you increase the remaining number of fractions to 42. To maintain the original probability of normal tissue complications, what should be the dose per fraction for the remaining treatment?
i. How will this affect tumor control probability, ignoring the overall treatment time?
j. If you continue with 1 fraction per weekday, your new treatment will extend the overall treatment time by 18 days. How will this affect tumor control probability?
k. How can you maintain both probability of tumor control and normal tissue toxicity?

6. A patient comes back for palliative treatment after having previously received a course of 39Gy in 30 fractions to the spinal cord. You want to give her 8Gy in a single fraction.
    a. Is this safe, assuming the previous treatment was recent and there has been no recovery?