Formation of discontinuous tumour implants at a distance from the main tumour mass
○ With two exceptions, all malignant tumours can metastasise --> gliomas and basal cell carcinomas
○ Approximately 30% of patients present with metastasis.

**Difference between metaplasia and dysplasia**

**Dysplasia**
- Disordered growth, limited to epithelium
- Loss of uniformity of individual cells
- Loss of their architectural orientation
- Normal --> dysplasia --> cancer
- Mild to moderate dysplasia may revert to normal
- High grade dysplasia = increased likelihood of progressing to cancer
- Severe dysplasia = carcinoma in situ = cells look like cancer cells but haven't invaded through the basement membrane yet

**Metaplasia**
- Substitution of one mature cell type for another mature cell type more suited to the environment
- The result of a chronic stimulus, when withdrawn may resolve to normal
- Is adaptive, not premalignant
- Physiological metaplasia: columnar epithelium turns squamous in the cervix (puberty/pregnancy)
- Pathological metaplasia: smoking causes metaplasia of columnar bronchial epithelium to squamous epithelium which can go on to become malignant. If smokers stop smoking, it's possible to reverse that metaplasia.

**Spread of tumours via blood vessels**
- Stephen Paget’s original seed and soil hypothesis proposed the organ preference patterns of tumour metastasis are the product of favourable interactions between metastatic tumour cells (the seed) and their organ microenvironment (the soil).
- The initial step of localised invasiveness enables in situ carcinoma cells to breach the basement membrane
- Thereafter, they may intravasate into either lymphatic or blood microvessels
- The latter may then transport these cancer cells to distant anatomical sites, where they may be trapped and subsequently extravasate and form dormant micrometastases
- Eventually some of the micrometastases may acquire the ability to colonise the tissue in which they have landed, enabling them to form a macroscopic metastasis.
- The last step – colonisation - seems to be the most inefficient of all
- The small probability of successfully completing all steps of this cascade explains the low likelihood that any single cancer cell leaving a primary tumour will succeed in becoming the founder of a distant macroscopic metastasis.

**Pathways of spread**

1. Direct seeding of body cavities and surfaces
   - Peritoneal
   - Pleural