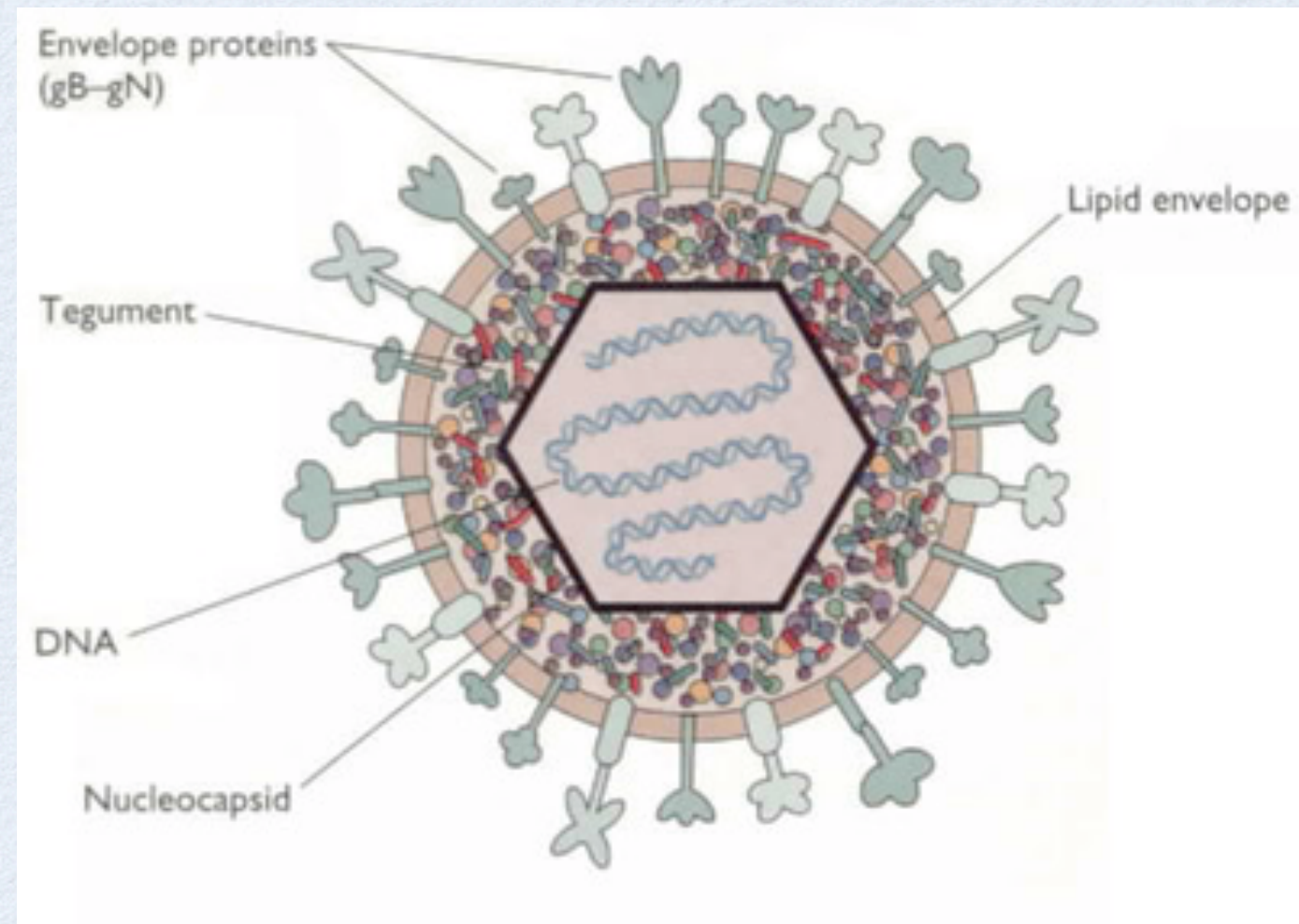


HERPES SIMPLEX VIRUS 1 (HSA-1)

FROM COLD SORES TO BRAIN LIQUEFACTION

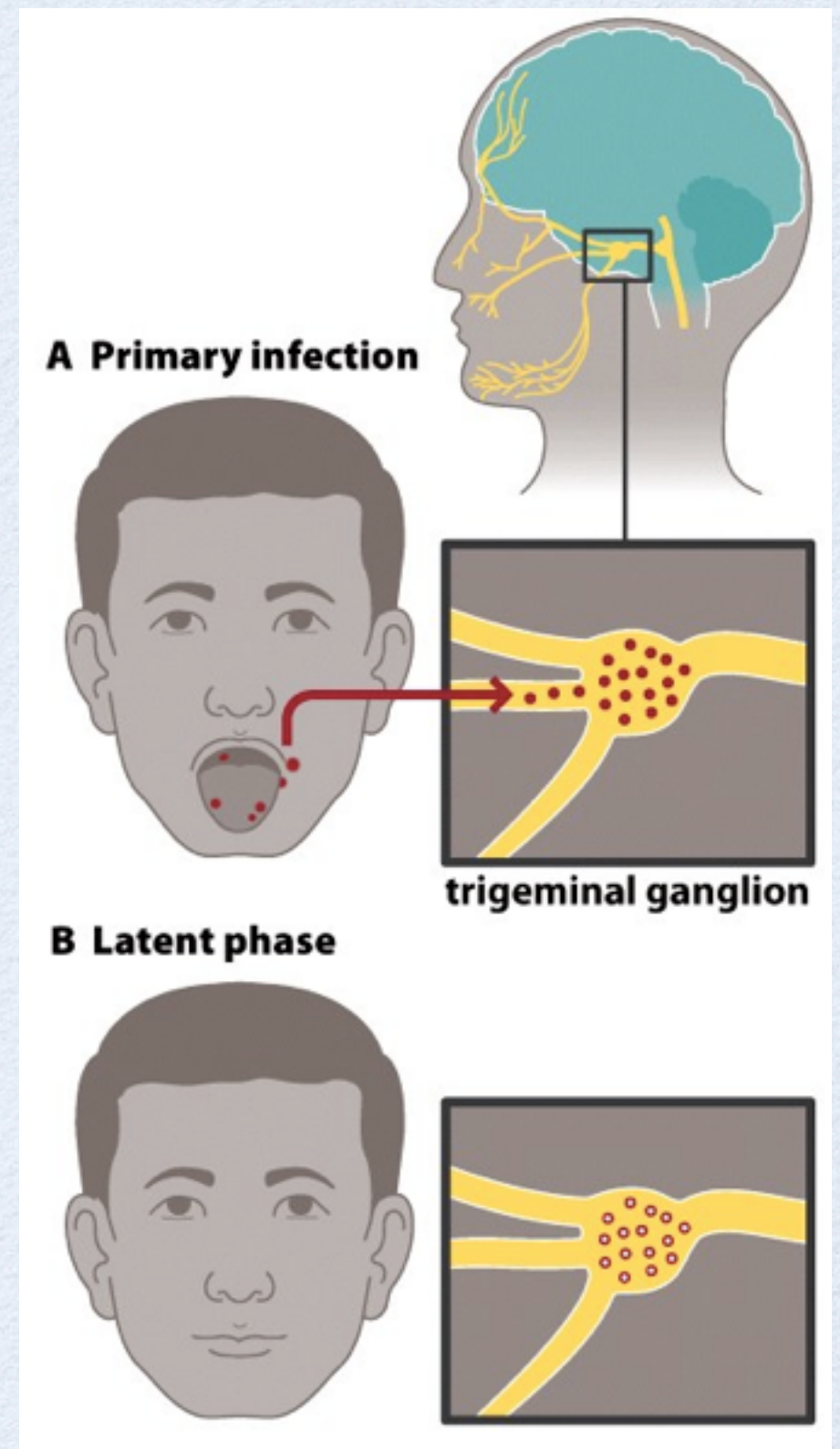
WHAT IS HSV-1

- HSV-1 is a herpesvirus, one of eight that cause disease in humans
- Herpesviruses have a double-stranded DNA genome in an icosahedral capsid surrounded by a lipid envelope



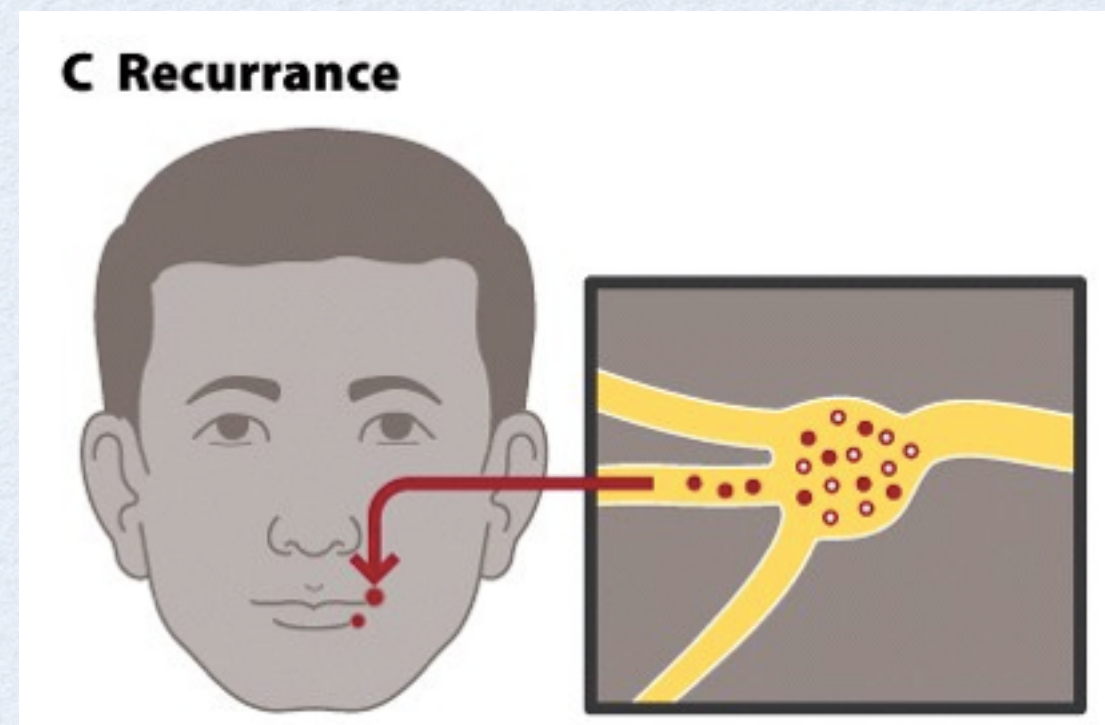
ENTRY AND SPREAD

- HSV enters through mucosal surfaces but not through intact skin
- HSV replicates within epithelial cells and spreads locally through budding from dying cells
- Virus can enter nerve cells and travel retrograde to nerve cell body



LATENCY AND REACTIVATION

- HSV is latent within nerves
- Reactivation can be triggered by local trauma, immunosuppression, UV light and “stress”
- Reactivation is most commonly asymptomatic and results infectious shedding of virus



HERPES SIMPLEX ENCEPHALITIS

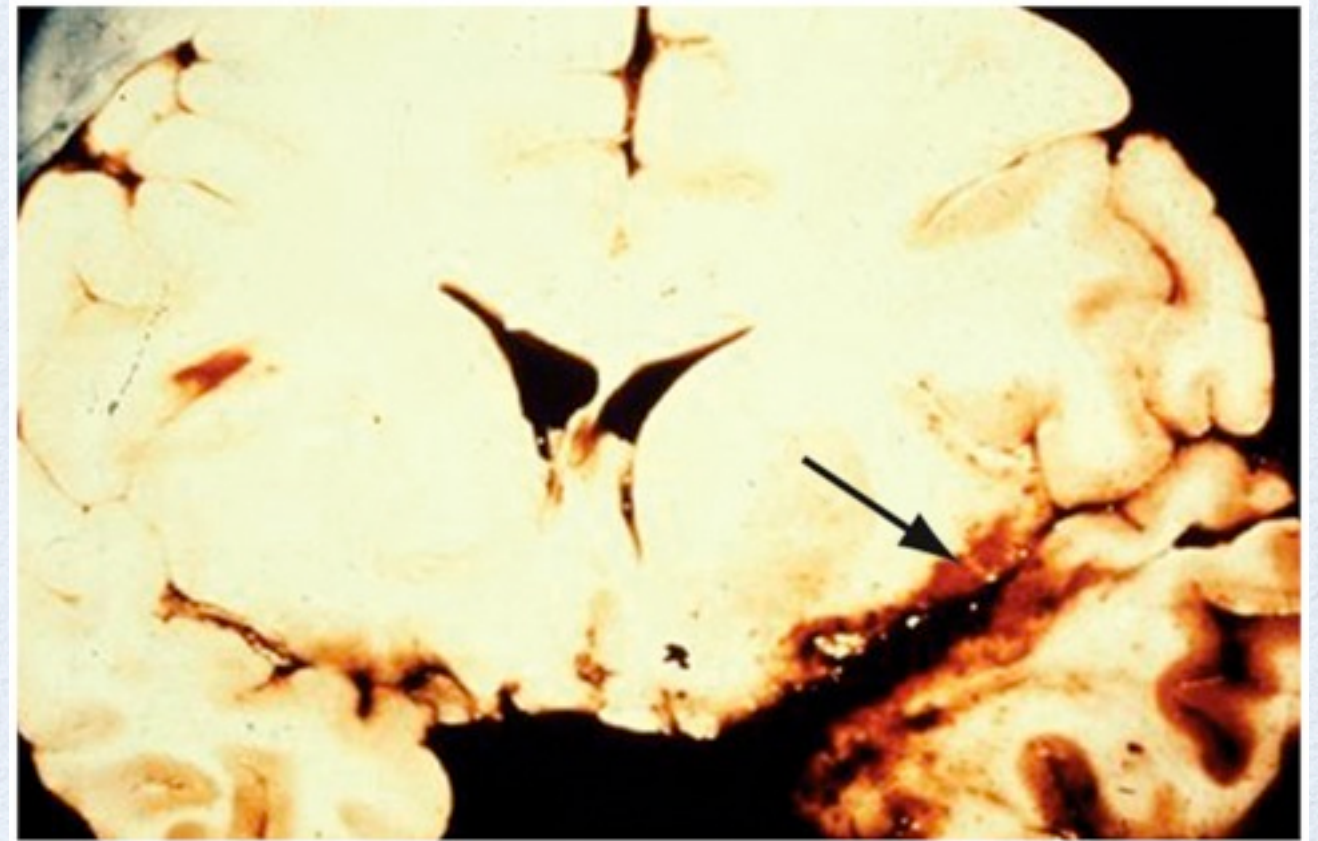
- UK incidence 1-3 per million population per year
- 90% of cases of encephalitis
- Two thirds are due to reactivation of latent virus
- 20-30% mortality with treatment
- 70% mortality untreated

CLINICAL PRESENTATION

- Non-specific, common symptoms and signs include altered behaviour, headache, fever, seizures and altered consciousness and less commonly vomiting, hemiparesis and memory loss
- PCR is most sensitive and specific diagnostic test but may be negative especially early in disease
- Neuroimaging (EEG, CT, MRI) shows focal rather than diffuse disease

PATHOGENESIS

- Route of entry into brain unclear
- Timing of entry into brain
- Hemorrhagic necrosis with liquefaction of affected brain tissue
- Acute inflammatory response characterized by influx of mononuclear cells



Case 14, Figure 3 Case Studies in Infectious Disease (© Garland Science)

TREATMENT

- No prophylactic vaccine
- High dose aciclovir for minimum 2 weeks
- Triple phosphorylation initiated by virally encoded kinase
- Immediate phosphorylation increases diffusion of drug into infected cells
- DNA chain terminator

