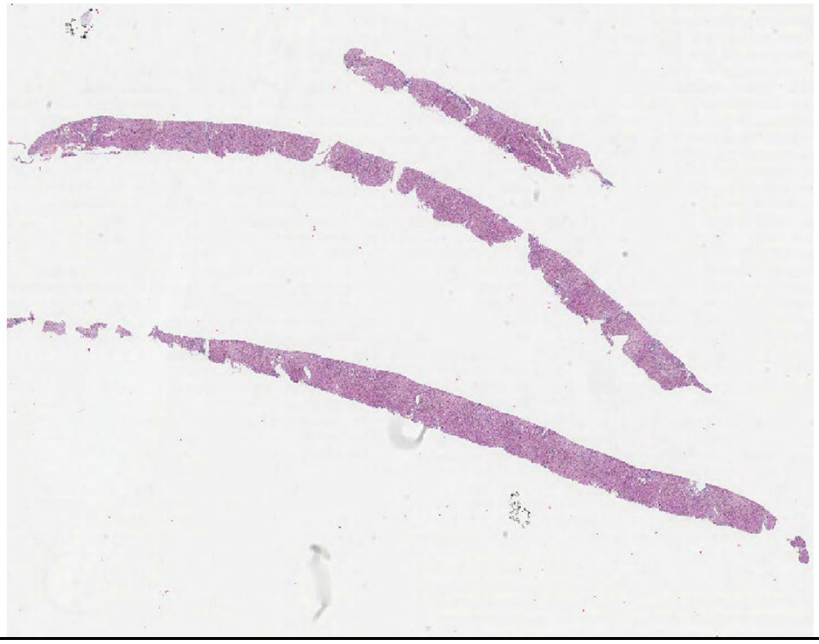
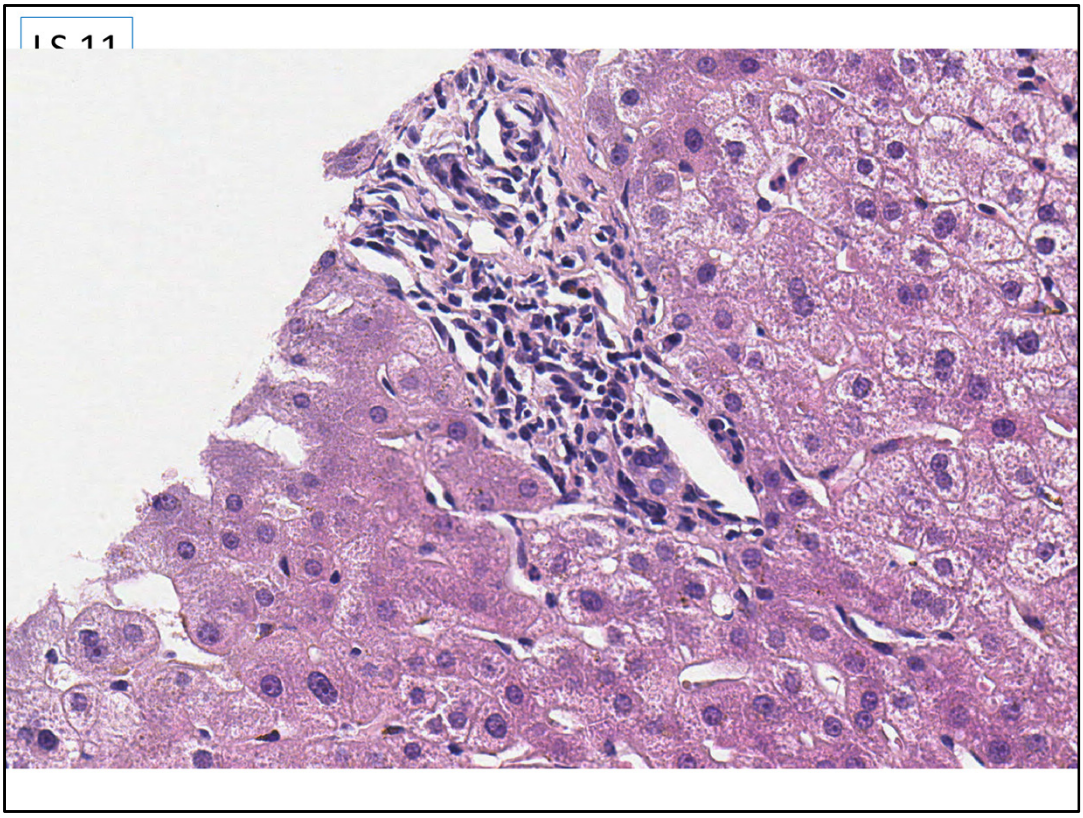
Case LS11 50 M

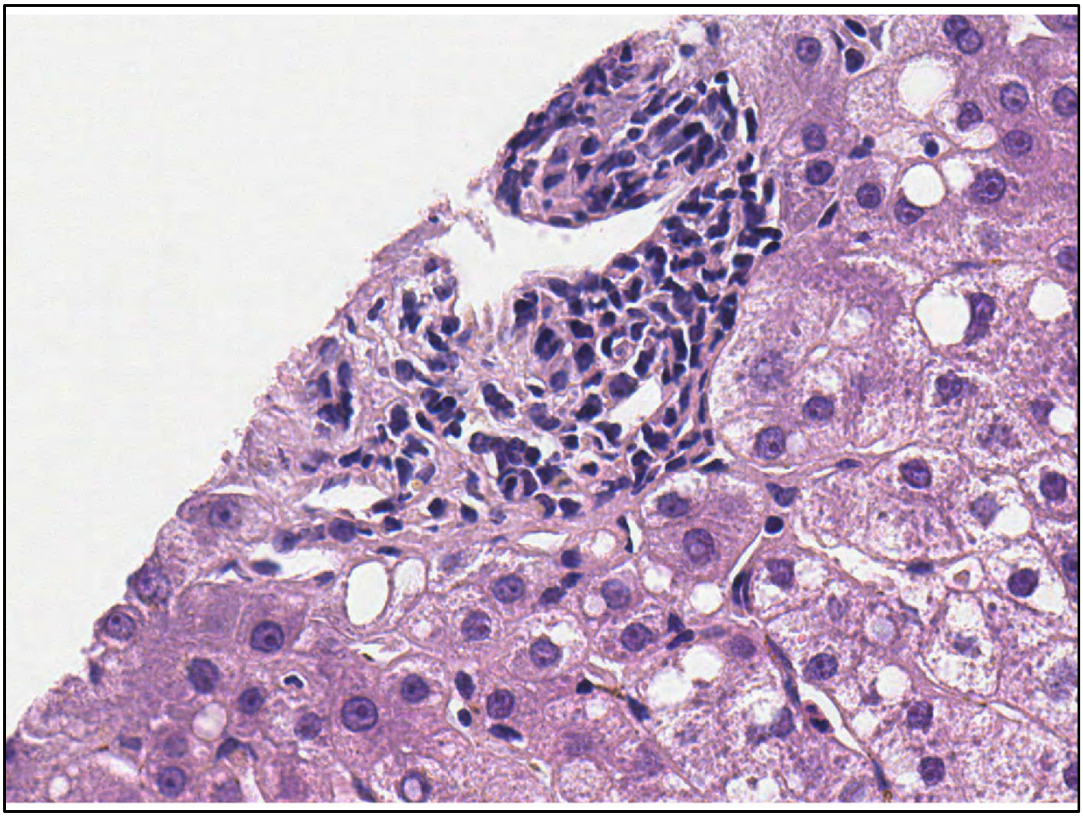
BMT for AML. Massively deranged LFTs, ALT 600

? GVHD, ? other liver pathology

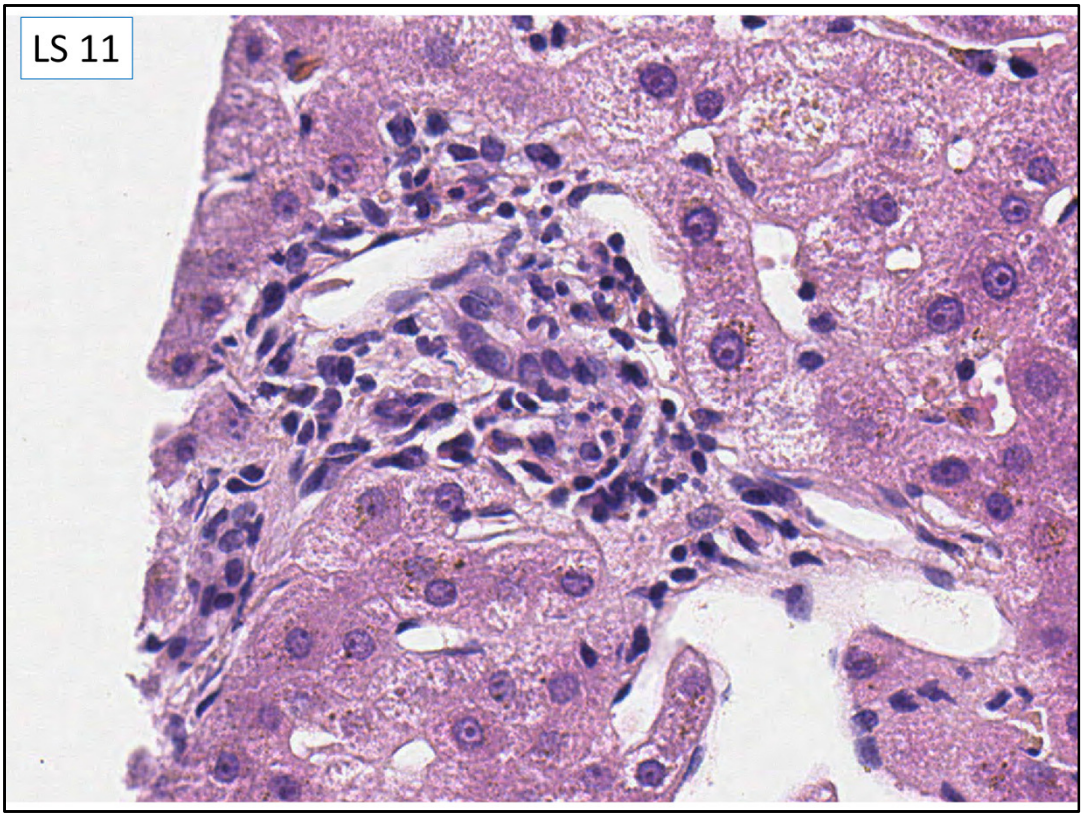




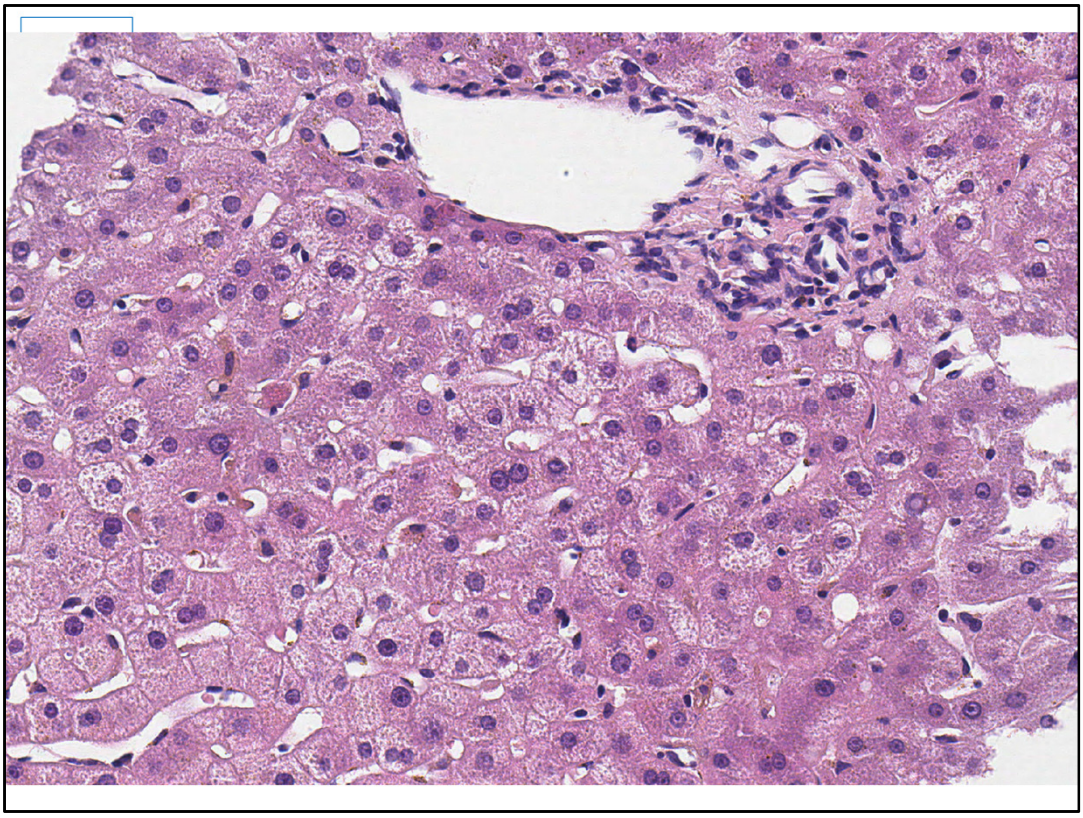
Despite the very high ALT of 600, > x10 the upper limit of normal, there is surprisingly little abnormality in the biopsy. The portal tracts show a slight increase in mononuclear cells and bile ducts are small with slightly irregular epithelium.



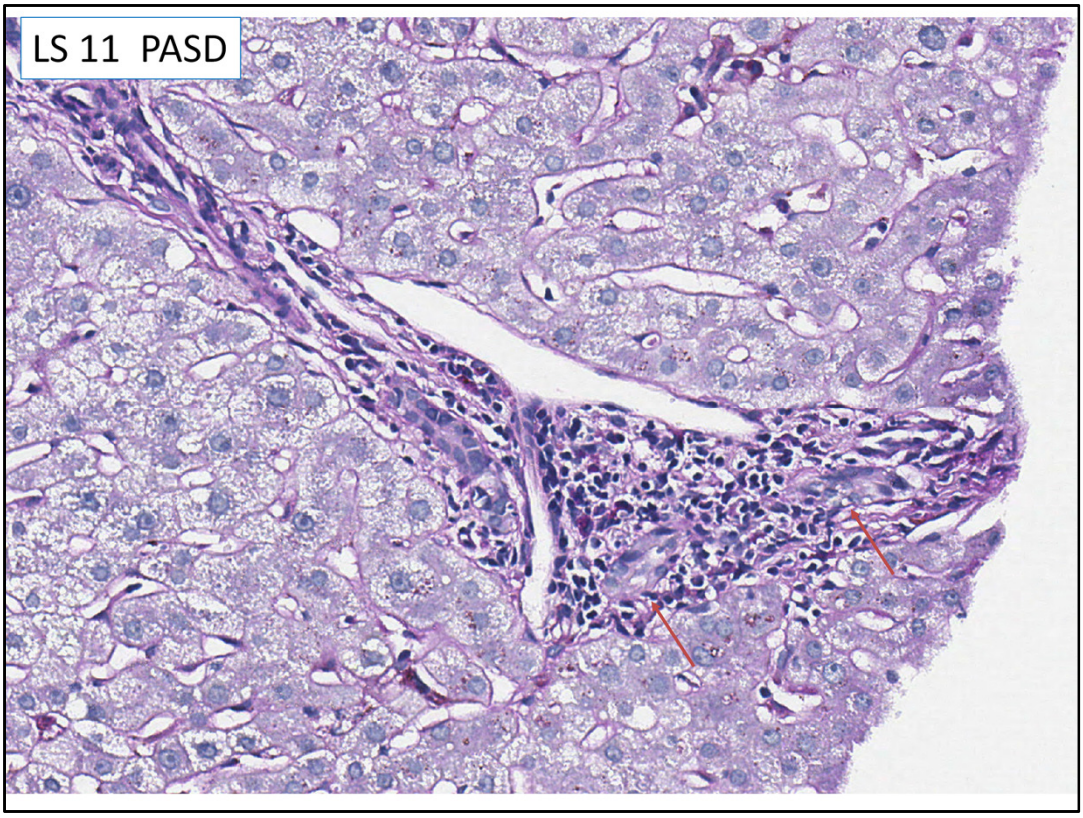
Another small portal tract with similar features .



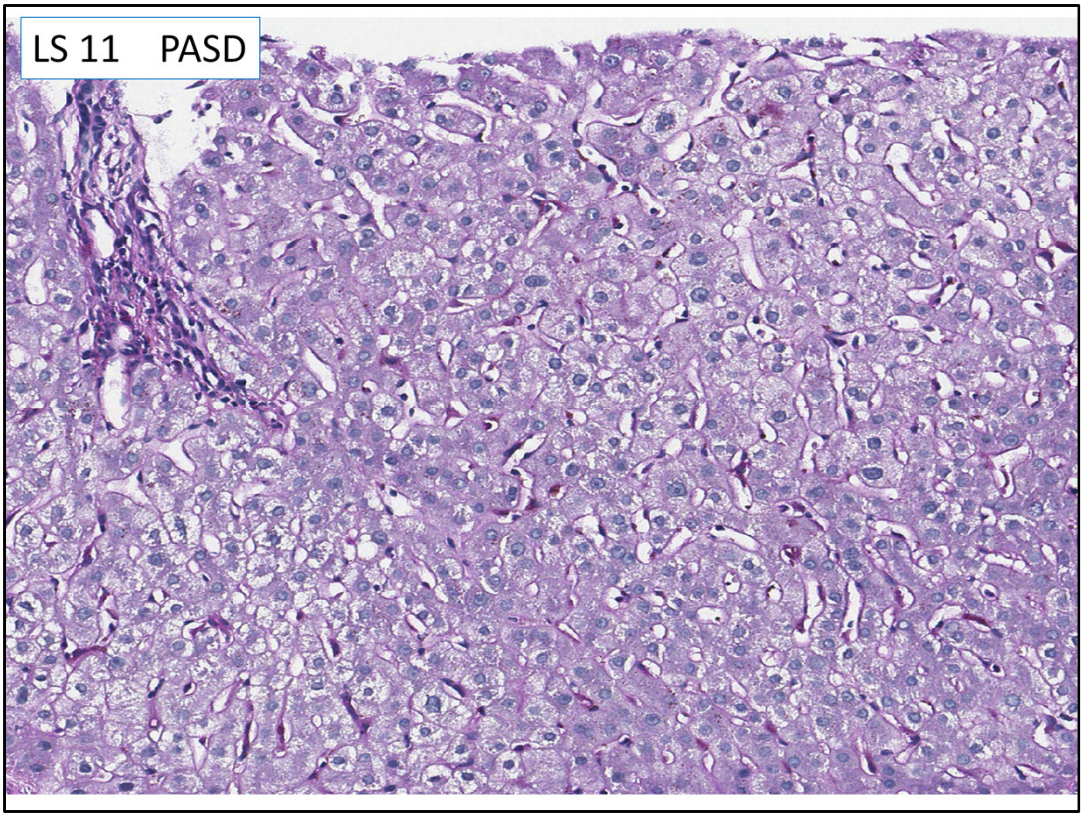
And another, the small duct has a gap in its ring of epithelial cells.



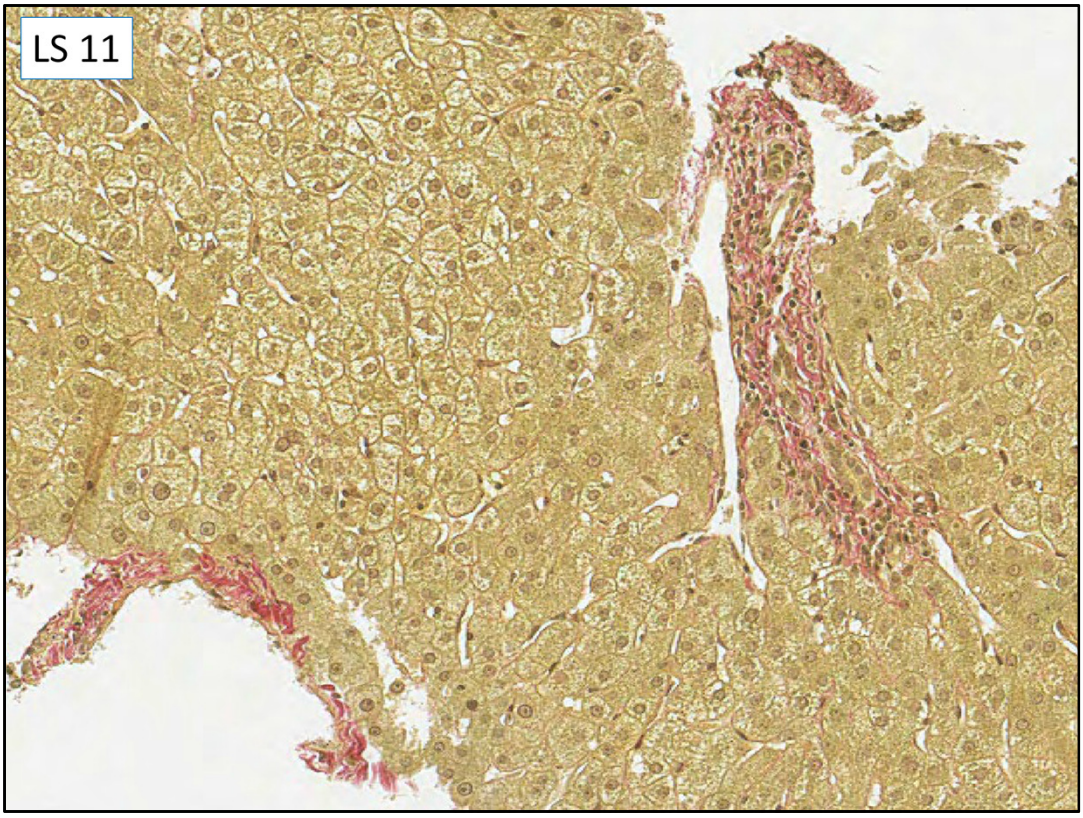
In the parenchyma there are occasional acidophil bodies, a hint of cholestasis, but in general surprisingly little abnormality. There is not a noticeable lobular hepatitis. There is not sinusoidal obstruction/evidence of veno-occlusive disease which may be associated with treatment of haematological malignancy, or of opportunistic infection such as CMV hepatitis.



Bile ducts are often easier to evaluate on the PASD stain than the H&E. In this portal tracts to atrophic, senescent morphology of the duct epithelium can be seen (arrows).



There is only a hint of Kupffer cell hyperplasia on the PASD stain. Overall, it is the absence of parenchymal injury – no alternative cause for the very high ALT – which is important in supporting the diagnosis of 'consistent with GVHD'.



There is no fibrosis.

Case LS11 50 M

MBT for AML 11.1.18. Massively deranged LFTs, ALT 600
? GVHD, ? other liver pathology

A	Resolving hepatitis ? EBV related
B	Suspicious of veno-occlusive disease
C	Consistent with graft v. host disease
D	Non-specific reactive changes
E	Infiltration by AML

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Correct response: C consistent with graft v host disease
The abnormalities in the biopsy can be very subtle in comparison with the marked abnormality of liver enzymes. The mild abnormalities of the bile ducts are important, and may be easier to see on the PASD stain. In this case, the diagnosis was corroborated by the clinical features and response to treatment. More advanced GVHD is associated with loss of bile ducts and canalicular cholestasis. Some patients have a 'hepatitic' pattern with sinusoidal inflammation especially around hepatic venules ('perivenulitis'). Both patterns have counterparts in liver transplant pathology.

Comments on other options:
A: resolving hepatitis ? EBV related – this would show evidence of previous lobular hepatitis, including PASD positive Kupffer cells as a sign of previous hepatocyte injury. EBV infection classically has plentiful sinusoidal lymphocytes. The features are not present here.

B: suspicious of veno-occlusive disease – VOD is often a clinical diagnosis in patients following treatment for haematological malignancy, not often biopsied. The biopsy would show congestion around terminal hepatic venules, and the van Gieson stain would show narrowing or occlusion of these veins. Like GVHD, the abnormalities can be subtle in relation to the degree of clinical abnormality.

D: non-specific reactive changes – is the term used for minor abnormalities, e.g. slight increase in inflammatory cells in a minority of the portal tracts, and/or mild lobular disarray or Kupffer cell hyperplasia. This is preferable to 'non-specific hepatitis' which

implies a primary liver disorder, whereas nonspecific reactive changes can be seen in systemic illness, with the liver affected as a ‘bystander’.

E: infiltration by AML – look in sinusoids for liver infiltration in haematological disorders, either leukaemic infiltrate, or in other context extra-medullary haematopoiesis., if there are increased sinusoidal cells without hepatocyte injury. This is one of the clinical diagnoses to consider, and comment on the absence of evidence in the report.