This type of reaction should be demonstrable histologically by muscular, glandular and secretory changes identified by microscopy.

3. If ammonia is present in excess in the blood as a proximate cause of death, this should be demonstrable in blood samples and vitreous fluid, and there is no evidence for this.
4. The liver in SIDS cases shows no abnormality and had it been acutely affected by an influx of ammonia, there should be changes.
5. Ammonia in excess leads to cerebral changes of an acute type and none have been demonstrated.
6. If the ammonia is postulated as a cause of petechiae in the lungs due to local damage, this does not account for the presence of petechiae in the thymus and pericardium.

There is evidence to explain how risk factors could contribute to susceptibility of infants to infectious agents to triggering the series of events leading to SIDS[17]; however, that presented for *H pylori* needs to be substantiated by more than one method and testable hypotheses proposed to explain how these bacteria might contribute to the series of events that lead to SIDS.

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Controls not matched

EDITOR,—The paper by Kerr *et al* reported an association between *H pylori* and sudden infant death syndrome (SIDS). We have reviewed their data and believe that the methods used may have led to incorrect conclusions.

Kerr *et al* examined retrospective material from 32 cases of SIDS infants and 8 non-SIDS controls. They used nested PCR followed by an ELISA detection step which would have made their method exquisitely sensitive. Consistent with this, no other method was able to confirm that *H pylori* was actually present. Instead, Kerr *et al* used a relative increase of "*H pylori* signal" above that of the mean +2SD for a control group, as an indicator of *H pylori* presence. This prompted us to more carefully consider the appropriateness of their control and patient groups.

Since ethnicity and socioeconomic details of the SIDS infants were not given, we could not confirm that these matched the control infants. We also noted important clinical details of the controls which could make them inappropriate. It appears that most of the controls would have had very little bacterial contamination of the PCR specimens because they died in hospital while on antibiotic therapy for sepsis, or were deceased very soon after premature birth. In addition, they might have been transferred to refrigeration very soon after death. SIDS infants however, probably died at home, many hours before being refrigerated.

Finally, as *H pylori* is a gastric organism, it was surprising to find the bacterium in lung or trachea of eight patients (ureC gene) or six patients (cagA gene) in whom gastric specimens were negative.

Since Kerr's paper was widely reported in the media, we believe that it needs to be stated that the case for *H pylori* as a cause of SIDS is certainly unproven and is in quite considerable doubt.

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No association in a Chinese population

EDITOR,—We read with great interest the paper by *Kerr et al* on the association between *H pylori* infection and SIDS. However, we cannot agree with the speculation the authors made.

Recently, we performed a similar retrospective analysis of nine cases of SIDS and eight controls collected in our hospital over the past two years. Controls were selected from infants with known cause of death, including congenital malformation, infection, metabolic disease, and drug intoxication (see table).

Table 1 Characteristics of SIDS cases and controls

	Sex	Age	Diagnosis
Controls			
1	F	4 months	Congenital heart disease
2	F	2 months	Morphione toxicity
3	M	13 hours	Bronchopneumonia
4	M	1 hour	Amniotic fluid aspiration
5	M	6 months	Premature, septicemia
6	M	3 months	Congenital brain tumour
7	M	6 months	Glutaric aciduria type I
8	M	2 months	Extreme premature
Cases			
1	M	3 months	SIDS
2	M	3 months	SIDS
3	M	13 months	SIDS
4	M	7 days	SIDS
5	M	5 days	SIDS
6	F	8 months	SIDS
7	F	2 months	SIDS
8	F	2.5 months	SIDS
9	M	2 months	SIDS

The formalin-fixed and paraffin-embedded stomach, trachea, and lung specimens obtained during postmortem examination were retrieved. Initial histological examination was performed by an experienced pathologist to look for any evidence of *H pylori* colonisation in these specimens. In addition, we used three different PCR assays that amplify two regions of the ureB gene[1 2] and the cagA gene[3] to detect the presence of *H pylori* DNA in these samples.

Histological examination failed to show any *Helicobacter* like organism in these samples. Moreover, despite using three different sensitive PCR assays, we failed to show the presence of *H pylori* DNA in the stomach, lung, or trachea of the SIDS and control patients.

Viable *H pylori* has recently been recovered from the vomitus of infected children and adults.[2] Conceivably, it could lead to silent aspiration of gastric contents into the lung and result in bronchopneumonia. However, the failure to detect the organism in the stomach, trachea, and lung specimens, together with the absence of features to suggest aspiration pneumonia as the cause of death in these infants, argue against the validity of this speculation. With the high prevalence of *H pylori* infection in Chinese, one would expect a parallel high incidence of SIDS in our ethnic group, which does not fit into any epidemiological observations. Taken together, the significance of *H pylori* as a cause of SIDS is highly questionable.

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