presence of procoagulant factors (macrophages and activated leucocytes are known to increase activation of thrombin and fibrin formation) and co-existence of sarcoidosis and antiphospholipid syndrome.[5] Antiphospholipid antibodies occur in 2–5% of the general population, but in up to 38% patients with sarcoidosis, correlating with poorer prognosis.[5]

Overall, our findings would be viewed as hypothesis generating, providing a platform for further study, and supportive of the anecdotal observations made in our Sarcoidosis Clinic. Despite the limitations discussed above, PE should be considered in patients with sarcoidosis when there is sudden deterioration in dyspnoea.

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► Additional data are published online only. To view these files please visit the journal online (http://thorax.bmj.com).

Funding The data set of the Oxford Record Linkage Study was funded by the former Oxford Regional Health Authority and, over many years, was built by Leicester Gill and Glenys Bettley. The NIHR Co-ordinating Centre for Research Capacity Development funds the Unit of Health-Care Epidemiology to undertake research using the linked data set.

Competing interests None.

Ethics approval This study was conducted with the approval of the Oxfordshire Ethics Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

Accepted 9 March 2010
Published Online First 13 September 2010

Thorax 2011;**66**:447–448.
doi:10.1136/thx.2010.134429

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Chemotherapy should not be withheld from patients with an indwelling pleural catheter for malignant pleural effusion

A 1–12% rate of pleural infection has been observed in patients with an indwelling pleural catheter (IPC) to manage malignant pleural effusion (MPE), leading to concern that systemic chemotherapy may increase infection risk.[1–5] This study aimed to determine whether chemotherapy increases the infection rate in patients with an IPC.

Data were collected from a prospectively maintained database, hospital notes and electronic records in a tertiary centre. All patients who had an IPC inserted between May 2006 and January 2010 to treat an MPE without pleural infection at the time of insertion were included. Pleural infection was defined as satisfying all of the following criteria: (1) positive pleural fluid culture; (2) symptoms of infection; and (3) treatment with antibiotics.

Eighty-two IPC placements in 78 patients with an MPE were included (table 1). Malignancies included breast cancer (n=21), mesothelioma (n=18), non-small cell lung cancer (n=13) and adenocarcinoma of unknown origin (n=8). Of 44 patients who received systemic chemotherapy (including cytotoxic chemotherapy and targeted therapies), 23 had an IPC during chemotherapy (table 1) (see online supplement for details of chemotherapy regimens). On average, patients had 2.5 cycles of chemotherapy with an IPC present (range 1–7 cycles). None of these 23 patients had WHO grade III or IV toxicities. Ten patients developed neutropenia at some point during their chemotherapy, of whom three had an IPC present at that time. In all cases neutropenia lasted <1 week and none of these patients developed infections.

Seven patients (9%) developed pleural infections, only one of whom was receiving chemotherapy (sunitinib) during the time the IPC was present. There was no difference in infection rate between those who received chemotherapy while the IPC was present and those who did not (Fisher exact test, p=0.667). Of the other six, three had previously received chemotherapy, one had chemotherapy after treatment of the infection and two never received chemotherapy.

Nine (11%) other patients had a positive pleural fluid culture (four during chemotherapy) without symptoms of infection and not requiring antibiotics. Five of these patients had subsequent negative pleural fluid cultures without antibiotic treatment. This may have been due to colonisation or contamination.

A range of organisms were identified, with *Staphlococcous aureus* the most common in cases of infection (three cases of infection, one of colonisation) and coagulase-negative *Staphlococcus* the most common in colonisation (one infection, five colonisation).

The median time the IPC was present was 71 days (range: 6–711). Twenty-nine IPCs (35%) were removed prior to death. Although the no chemotherapy group appear to have a shorter IPC duration than the chemotherapy group, this was because of a higher mortality in the no chemotherapy group. The median time from IPC insertion to infection was 103 days (range 40–206). In the three patients who developed infection following previous chemotherapy, the time from last dose of chemotherapy to infection was 301 days (range 90–639).

These results show that systemic chemotherapy did not increase risk of pleural infection in this cohort of patients with IPCs. We conclude that an IPC is not a contraindication to chemotherapy.

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Table 1 Patient demographics

	Systemic chemotherapy while IPC in situ	No systemic chemotherapy while IPC in situ
Number	23	59
Mean age (years)	60	67
Males:females	11:12	34:25
Median duration IPC was in situ (days) (range)	84 (11–487)	66 (6–711)
Median duration IPC was in situ in patients in whom IPC was removed prior to death	84 (11–239)	82 (30–711)
No. of patients who died with IPC in situ (%)	13 (57%)	40 (68%)
Average time patient received chemotherapy while IPC was in situ (days) (range)	68 (11–208) (~2.5 cycles)	–
Pleural infections	1 (4%)	6 (10%)
Colonisation of IPC	4 (17%)	5 (8%)

IPC, indwelling pleural catheter.

► Additional materials are published online only. To view these files please visit the journal online (http://thorax.bmj.com).

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

Accepted 23 March 2010
Published Online First 29 September 2010

Thorax 2011;**66**:448–449.
doi:10.1136/thx.2009.133504

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CORRESPONDENCE

Television viewing and asthma: spurious relationship?

In the April 2009 issue of *Thorax*, Sherriff and co-authors report on data taken from the ALSPAC study, addressing the association between television viewing in early childhood and the development of asthma.[1] They found that, after adjustment for body mass index, there was a relationship between the two, showing a significant trend.

I was surprised to see that television viewing was viewed solely as a proxy for a sedentary lifestyle, but not as being associated with other risk factors for developing asthma. For example, although the authors corrected for smoking during pregnancy, they did not include parental smoking at home in their model. It is not unlikely that among parents of children that were reported to have been watching television for longer, many of them were smoking in the presence of their child.

Adjustment for such additional factors is warranted before discussing the consequences of the study findings.

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Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

Accepted 21 August 2009
Published Online First 23 August 2010

Thorax 2011;**66**:449. doi:10.1136/thx.2009.121111

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Author's reply

We thank Dr van der Wouden[1] for his interest in our paper.[2] He raises the question as to whether our observations could be mediated (confounded) or modified by contemporaneous environmental tobacco smoke (ETS) exposure during periods of TV watching.

We did find that parents of children with longer reported TV watching were more likely to report that the child was exposed to tobacco smoke: 17.4% of children watching no TV at all were exposed to postnatal ETS, 25% of children watching less than 1 h per day, 33.1% of children watching 1–2 h per day and 42.3% of children watching 2 h or more per day (p linear <0.001). However, only 6.4% of children exposed to postnatal ETS reported asthma at 11.5 years compared with 5.9% not exposed (p for difference between proportions 0.62).

Therefore, despite the association of ETS exposure with reported TV viewing, the lack of a strong association of ETS with asthma at 11.5 years in children asymptomatic up to 3.5 years made it unlikely that postnatal ETS had an independent effect on asthma development in this sample.

In our paper, we chose to adjust the final model for prenatal tobacco smoke exposure only. This was chosen because there was a high degree of co-linearity between prenatal and postnatal smoking in this population and prenatal exposure has been reported to be more strongly associated with asthma in several studies (see the recent meta-analysis by Pattenden *et al*).[3] We have previously reported that prenatal exposure is associated with early onset wheezing,[4] but that neither prenatal nor postnatal exposure to ETS were associated with later onset or persistent wheezing, more likely to be phenotypes associated with asthma. By excluding children who wheezed at any time before 3.5 years from our study, we think it is likely that we have attenuated any potential effect of early smoke exposure on the outcome. Finally, when we considered reported postnatal ETS as a covariate in our final model along with prenatal exposure, we found no attenuation of the association of TV viewing with asthma.

We also considered the possibility that postnatal ETS may have modified the association of prolonged TV viewing with asthma at 11.5 years, as suggested by the correspondent, but a formal test of interaction between TV viewing and ETS on asthma outcome did not support this (p=0.78). Asthma prevalence at 11.5 years stratified by postnatal ETS exposure is shown in table 1.

Table 1 Asthma prevalence at 11.5 years stratified by postnatal ETS exposure

% (n) with asthma at 11.5 years		
TV viewing	**Not exposed**	**Exposed**
Not at all	4.3 (3/70)	10 (1/10)
<1 h per day	4.5 (30/663)	3.2 (6/187)
1–2 h per day	5.5 (52/952)	5.9 (26/442)
2+ h per day	9.1 (39/429)	8.8 (22/249)

ETS, environmental tobacco smoke.

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Provenance and peer review Not commissioned; not externally peer reviewed.

Accepted 24 August 2010
Published Online First 27 January 2011

Thorax 2011;**66**:449. doi:10.1136/thx.2010.149740

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Steroid-induced hyperglycaemia and pulmonary disease

Chakrabarti and colleagues recently reported that hyperglycaemia within 24 h of admission could be used as a predictor of outcome during non-invasive ventilation in decompensated chronic obstructive pulmonary disease (COPD).[1] Hyperglycaemia was unrelated to prior oral corticosteroid use in this study, but duration of steroid preceding admission was not reported. Furthermore, as this group included only 18 patients it would be insufficiently powered to detect a modest rise in glucose. Doctors are vigilant to the occasional patient who develops symptomatic hyperglycaemia whilst taking steroids, but are less attentive to small changes in glucose. Li *et al* recorded complications of steroid treatment in a cohort of 1291 patients with SARS (severe acute respiratory