uterotonic drugs compared with 43.1 among those not exposed. From a multiple linear regression with all above mentioned confounders included we estimated the difference in Boerge Prien score to be −0.58 (95% confidence interval −1.25 to 0.08) between those exposed and not exposed to uterotonic drugs.

## Comment

Friedman et al examined 156 children 23 to 62 months after births associated with spontaneous labour, labour induced with oxytocin, or labour induced with dinoprostone. The prevalence of neurological or developmental abnormalities not attributable to events after delivery was the same overall in induced and spontaneous labours, but those abnormalities occurring after induction of labour all followed use of oxytocin.[5] Our data indicate that exposure to uterotonic drugs does not substantially affect cognitive function 20 years later. A small difference due to non-differential misclassification, however, cannot be ruled out. A strength of our study is the large size, the population based design, and complete ascertainment. It is unlikely that selection bias and confounding explain the lack of association.

Contributors: HTS, KJR, and MWG in collaboration with SS initiated the establishment and design of the cohort. HTS initiated this study and discussed the hypothesis with KJR. HTS and FHS analysed the data. PF collected the outcome data. SS linked the data to the birth registry. All authors participated in the interpretation of the findings. HTS, KJR and FHS wrote the first draft of the paper. All participated in editing the paper. HTS is guarantor of the paper.

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# Giant cell arteritis and thyroid dysfunction: multicentre case-control study

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website *extra*

*An additional table appears on our website*

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The association between giant cell arteritis and thyroid dysfunction remains controversial, but as giant cells are a possible feature of Graves' disease, a common pathway has been suggested. In two series of 101 and 98 patients, the prevalence of hyperthyroidism was reported to be six times higher in cases of giant cell arteritis than in controls.[1 2] This was not confirmed on smaller series,[3 4] but 15 cases of hypothyroidism were reported in 31 patients with giant cell arteritis.[5]

We conducted a multicentre case-control study on cases of giant cell arteritis to investigate this relation.

## Subjects, methods, and results

Assuming a prevalence of thyroid dysfunction of 1% in the general population and an odds ratio of 6 for hyperthyroidism in the patient group, the sample size requested, with $\alpha = 0.05$ and $\beta = 0.2$, had been estimated to be 269 cases and controls.[2]

We prospectively studied 285 cases of giant cell arteritis (205 women, mean age 74.7 ± 8.2 years; 80 men, 72.7 ± 8.2) newly diagnosed during 1991-96. An experienced pathologist reviewed 262 (92%) of the biopsies: temporal arteritis was confirmed in 145—68 were classed as negative (eight did not have a biopsy), and 72 were classed as having polymyalgia rheumatica alone (22 did not have a biopsy). Blood samples taken up to 48 hours after diagnosis were sent to a reference laboratory.

Controls, randomly selected by computer from residents of Saint-Etienne affiliated to a health insurance company, were matched to cases for age and sex. Of the 222 controls participating, 208 (94%) agreed to have a blood sample taken (140 women, mean age 74.9 ± 8.7 years; 68 men, 71.7 ± 8.0) (table). Neither cases nor controls had clinical signs or symptoms of thyroid dysfunction.

We measured concentrations of free thyroxine, thyroid stimulating hormone, and antithyroid peroxidase antibodies by standard radioimmunoassays. Antithyroglobulin antibodies were measured as follows: sera were incubated at room temperature with thyroglobulin labelled with 125-iodine, and the immune complexes were precipitated in fetal veal buffer with polyethylene glycol. A positivity threshhold of 50 U/l for a population free of thyroid disease was determined.

We performed multiple logistic regression. Dependent variables were high and low concentrations of thyroid stimulating hormone, high and low concentrations of free thyroxine, and concentrations of positive or negative antiperoxidase antibodies, positive or negative antithyroglobulin antibodies, and positive or negative antithyroid antibodies (antiperoxidase or antithyroglobulin). Independent variables were case or control, geographical origin (north or south), age, sex, and clinical subgroup of patients.

When we took potential confounders into account, we found no difference between cases and controls.

Thyroid status of cases of giant cell arteritis and controls. *Values are numbers (percentages) unless stated otherwise

| Variable | Cases (n=285) | Controls (n=208) | P value | Odds ratio (95% CI) |
|---|---|---|---|---|
| **Thyroid stimulating hormone†** | | | | |
| Low concentration | 13 (4.6) | 5 (2.4) | 0.171 | 3.06 (0.62 to 15.20) |
| High concentration | 12 (4.2) | 16 (7.7) | 0.227 | 0.38 (0.11 to 1.82) |
| **Thyroxine‡** | | | | |
| High concentration | 2 (0.7) | 1 (0.5) | 0.546 | 0.34 (0.01 to 89.62) |
| Low concentration | 7 (2.46) | 0 | 0.324 | NA |
| **Antibodies** | | | | |
| Antiperoxidase | 42 (14.7) | 29 (14.0) | 0.327 | 1.57 (0.63 to 3.89) |
| Antithyroglobulin | 71 (24.9) | 47 (22.6) | 0.574 | 1.26 (0.59 to 2.66) |
| Positive antiperoxidase and antithyroglobulin | 89 (31.2) | 59 (28.4) | 0.3145 | 1.42 (0.71 to 2.84) |

*Independent variables: case or control, sex, age, geographical origin (north or south), and clinical subgroup of patient (positive or negative biopsy for temporal arteritis or polymyalgia rheumatica).
†Normal range 0.2-4 mIU/l. ‡Normal range 10-26 pmoles/l.

Antithyroid antibodies occurred more frequently in women than in men, and prevalence increased with age.

We found no difference between cases and controls when thyroxine or thyroid stimulating hormone titres were outside the normal range, or when antithyroglobulin or antiperoxidase antibody titres were positive (Wilcoxon sum rank test) (table on website).

## Comment

The prevalence of high concentrations of thyroid stimulating hormone and antithyroid antibody was similar in cases and controls at the onset of the disease. After adjustment for potential confounders, we found a threefold but non-significant increase in the risk of hyperthyroidism in cases when thyroid stimulating hormone concentrations were measured. If the risk was to be significant a sample size of 641 patients and 2564 controls would be needed; such a sample size with incident cases of giant cell arteritis seems unrealistic. However, a common pathway for Graves' disease or hypothyroidism and giant cell arteritis seems unlikely. Determination of free thyroxine concentrations is probably less reliable in inflammatory syndromes, as thyroxine is bound to sera proteins.

The high prevalence of antithyroid antibodies in the controls should make researchers cautious when describing an association between autoimmune or inflammatory diseases and thyroid dysfunction in elderly patients.

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*A dilemma*

## A half mixed compound?

Some lessons learnt during high school chemistry remain with me, none more so than the distinction between a mixture and a compound:

Mixture: a substance consisting of two or more substances mixed together without any chemical bonding between them.

Compound: a substance that contains atoms of two or more chemical elements held together by chemical bonds.

As a teenager I did not object to being termed "half caste." For the past few years I have preferred, and asked others to use, "mixed race." Lately, I have been aware of the inadequacies of this phrase, and now consider "compound ethnicity" to be more appropriate.

My father is of Asian and my mother of European ethnic origin. We do not, nor do the cultures of which we are a part, adhere to a caste system, hence I cannot be "half caste." It is also a derogatory term—suggesting that I do not belong to either of my parent's groupings; that I am neither here nor there. To continue this thought, any children my wife, Sally, and I produce would therefore be quarter caste in regard to Asian and three quarter caste with respect to European ethnic origin. As may be seen by observing any family over several generations, the extent to which individuals display, and to which they choose to follow, characteristics of their family varies greatly, and in an unpredictable manner.

Mixed race seemed a better classification; it seemed to have no disparaging connotations, nor any reference to caste. However, for some this gives a negative impression, as in "mixed up." In addition, if I am mixed race who can determine which parts of me are from my father and which from my mother? Is my left arm from one and my right from the other? Is my knee from Punjab and my elbow from Norfolk? In terms of character and of appearance—for example, skin colour—I am not a mixture, but a compound.

I believe the best description is to say that someone is of compound ethnicity. It has the additional benefit of necessitating the use of a noun to give the adjectival phrase meaning—for example, she is a woman of compound ethnicity; this boy has compound ethnicity. There may be future improvements to this term, but for the time being let us avoid the use of halving and mixing with reference to ethnicity and consider the basic distinction between a mixture and a compound.

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