perhaps be added, and a number of doses given after operation. This practice was followed by nearly a third of surgeons, although a similar proportion began antibiotic treatment only at the time of operation for a gangrenous or perforated appendix.

Applying povidone-iodine to the wound is a simple measure that confers some protection against wound infection and was used occasionally by nearly half of the surgeons in the survey. The available evidence is against peritoneal drainage (which was used by 77%), but a wound drain may be advantageous, although only 56% of the surgeons ever inserted one.

It is possible that topical agents and surgical drainage may be largely abandoned by those who are confident of the efficacy of the systemic antibiotic prophylaxis used.

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SHORT REPORTS

Papillary carcinoma of the thyroid in two brothers after chest fluoroscopy in childhood

Thyroid carcinoma rarely occurs in young men. The occurrence of papillary thyroid cancer in two brothers of similar age therefore suggested some common aetiological factor. A family history back to the grandparents and their siblings yielded no evidence of thyroid disease or of multiple polyposis. Both brothers, however, had been exposed to radiation from cardiac screening in childhood.

Case histories

Case 1—A 33-year-old man presented in 1973 with a painless nodule in the left lobe of the thyroid. He was euthyroid and otherwise fit. At operation a papillary carcinoma in the left lobe of the thyroid and an enlarged retro-clavicular lymph node containing carcinoma were removed. He was subsequently treated with ¹³¹I and remained well with no recurrence.

Case 2—A younger brother of case 1 was 38 when in 1979 he noticed a nodule in the left lobe of the thyroid. He was euthyroid and otherwise well. At operation a papillary carcinoma was found without evidence of spread beyond the thyroid. Near-total thyroidectomy was carried out followed by ¹³¹I treatment. He remained well without recurrence.

Sections from the thyroid tumours showed similar appearances (figure). Each was a well-differentiated thyroid papillary carcinoma, in places appear-

ing encapsulated. The adjacent thyroid tissue showed normal architecture and normal colloid-containing follicles. Inquiry disclosed that the two brothers had undergone detailed fluoroscopic examination of the heart at 11 and 9 years of age respectively because their mother had cardiac disease. The examinations had been carried out abroad, before image intensification was available, and no details of exact radiation exposure were available.

Comment

Familiar multiple polyposis (Gardner's syndrome) may be associated with the development of papillary thyroid carcinoma in siblings,¹ but this was excluded in our patients by the negative family history and absence of clinical features (appearances on sigmoidoscopy and barium enema were also normal). The only relevant history was the irradiation that each had received at fluoroscopy. Martin and Olson² concluded that irradiation of the thyroid during cardiac investigations is not negligible and may appreciably increase the risk of cancer.

The association of radiation with the development of thyroid cancer, often some 20-30 years later, is well recognised, though the dose has usually been in the range 2-5 Gy (200-300 rads).³ An increased incidence of cancer may, however, occur after much lower doses, and Pochin⁴ suggested an induction rate of 1% per Gy (1% per 100 rads). Using data on chest fluoroscopies⁵ as carried out at about the time that our patients were so investigated, we estimate that their thyroids had probably received a radiation dose of 0.2-0.3 Gy (20-30 rads). The eventual appearance of papillary cancers 22 and 28 years later confirms

the observations of Martin and Olson.² Other reports of radiation-induced thyroid cancer have dealt with the consequences of therapeutic irradiation. The history of past irradiation in our first patient was not obtained until his brother also developed a cancer, when

Histological appearances of well-differentiated papillary carcinoma of thyroid from two brothers. Above: case 1. Below: case 2. Haematoxylin and eosin $\times 100$ (original magnification).

inquiry was much more searching. Hence it is clearly important to pay careful attention to a history of special diagnostic procedures, particularly cardiac screening.

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Thromboxane A₂ in pregnancy and puerperium

A recent report presented evidence that despite the rise in the anti-aggregatory agent prostacyclin during late pregnancy the aggregation of platelets was enhanced.¹ An increase in the proaggregatory agent thromboxane A₂ (TxA₂) could explain this discrepancy.² To study the production of the proaggregatory and vasoconstricting agent thromboxane A₂ during human pregnancy and puerperium we measured the concentrations of its stable metabolite thromboxane B₂ (TxB₂) in plasma and serum from 45 women at 11-41 weeks of normal pregnancy, 11 puerperal women 53-60 days postpartum, and 22 healthy non-pregnant control women.

Subjects, methods, and results

Seventy-eight healthy women aged 17-39 volunteered for the study. None had taken drugs known to interfere with the synthesis of prostaglandins within 10 days of starting the study. We determined their TxA₂ production by measuring plasma and serum concentrations of its stable metabolite thromboxane B₂ (TxB₂) by radioimmunoassay. The rationale of this approach was to measure the circulating TxB₂ in vivo and the capacity of the platelets to produce TxB₂ during spontaneous clotting. The amount of TxB₂ produced during spontaneous clotting correlates closely ($r > 0.90$) with the amount of TxB₂ released from platelets during induced aggregation in platelet-rich plasma. Thus two blood samples were collected with the same venepuncture. To obtain plasma blood was taken into ice-cold heparinised tubes containing acetylsalicylic acid at a final concentration of 0.02 mmol/l (1.3 mg/100 ml) and centrifuged immediately at 4°C. For the sera, blood samples were taken into dry tubes and allowed to clot at 37°C for exactly 60 min before centrifugation. Both plasma and sera were stored frozen at -20°C until assayed. The samples from pregnant, puerperal, and non-pregnant women were equally distributed in different radioimmunoassay batches.

The results were subjected to the two-tailed Student's *t* test and regression analysis. TxB₂ concentrations in plasma were higher in the pregnant and puerperal women than in the controls, but no significant changes could be seen with advancing gestational age (table). Similarly, TxB₂ production during spontaneous clotting was greater during pregnancy and the puerperium than in the non-pregnant women. The TxB₂ concentrations in plasma and serum correlated significantly with each other ($r = 0.515$, $n = 78$, $p < 0.001$).

Mean ($\pm$ SE) concentrations of thromboxane B₂ in plasma and production of thromboxane B₂ during clotting of blood samples at 37°C for 60 min in pregnant and puerperal women compared with those in healthy, non-pregnant women

Population	No of women	TxB ₂ in plasma (pg/ml)	TxB ₂ in serum (ng/ml)
Pregnant women:	45	266.4 $\pm$ 38.6*	255.2 $\pm$ 14.1†
Pregnancy week 11-28	21	200.7 $\pm$ 47.1†	261.3 $\pm$ 19.4†
Pregnancy week 32-41	24	323.6 $\pm$ 58.6*	249.8 $\pm$ 20.7†
Puerperal women (53-60 days postpartum)	11	196.3 $\pm$ 23.7†	239.2 $\pm$ 17.7‡
Non-pregnant controls	22	99.3 $\pm$ 11.5	183.4 $\pm$ 14.5

* $p < 0.001$, † $p < 0.01$, ‡ $p < 0.05$ compared with concentrations in non-pregnant controls.

Comment

Our results show that both plasma TxB₂ concentrations and TxB₂ release in response to thrombin-induced platelet aggregation during spontaneous clotting rise during pregnancy and the puerperium. This rise in proaggregatory TxA₂ production could explain the increased platelet reactivity³ and the common occurrence of thromboembolic complications⁴ at these times. Our results could also explain why the capacity of platelets to aggregate was increased despite the enhanced production of the antiaggregatory agent prostacyclin in late pregnancy.¹ Conceivably the balance between TxA₂ and prostacyclin shifts to the side of TxA₂ dominance during pregnancy and puerperium.

The actual source of high plasma TxB₂ concentrations throughout normal pregnancy, as observed in this study, and at term⁵ is not known. Several pregnancy-associated tissues such as amnion, chorion, decidua, and placenta are capable of producing TxB₂ in vitro⁶; and they may contribute to the high plasma concentrations of TxB₂ during pregnancy but not during puerperium. Possibly the raised plasma TxB₂ concentrations reflect a greater incidence of micro-thrombi in the circulation at these times. But we must emphasise that despite the use of adequate anticoagulants and prostaglandin synthesis