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Relationship between Thyroid Function and Platelet Counts

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I read with interest the article on thrombocytope-nia in Graves' disease which appeared in Acta Haem-atologica 63:185-190 (1980). According to it, thyroid hormone stimulates the reticuloendothelial system (RES) resulting in thrombocytopenia. However, since I have not clinically experienced patients with hyper-thyroidism associated with thrombocytopenia except for the complication of idiopathic thrombocytopenic purpura [1] and have noticed the statement 'Platelets are normal in thyroid disease' in a classic endocrino-logical treatise [2], I tried to examine platelet counts in patients with both hyper- and hypothyroidism whom I myself had diagnosed and treated from 1981 to 1985, because I thought there was a great difference in the levels of thyroid hormones between these two conditions. I determined platelet counts by ELT-8 (Or-tho, USA) when I saw the patients for the first time and during their course, and used Student's t test by calculating the difference between matched pairs for statistical analysis. The platelet counts of 30 patients with hyper- and hypothyroidism are shown in table I. I could not discover any significant difference between the two conditions. When observed individually, platelet counts hardly changed following the change of thyroid function, but seemed invariable (table II). In addition, I noted the tendency for platelet counts to decrease with age. Thus, I think that, although it is true that thyroid hormone stimulates RES resulting in a shortening of platelet survival, the bone marrow function producing platelets is compensated for soon and marked thrombocytopenia does not appear. There is a study in which the increase of megakaryocytes in the bone marrow of patients with hyperthyroidism was reported [3]. It is said that the spleen is palpable in 30% of the patients with hyperthyroidism [4], but in Table I. Platelet counts in hyper- and hypothyroidism (1981-1985)

Hyper-	Sex	Age	Platelets	Hypothy-	Sex	Age	Platelets	thyroidism	($\times 10^4/\mu\text{l}$)	roidism

($\times 10^4/\mu\text{l}$)

1A.M.
F
28
28.3
1' K.T.
F
35
2H.T.
F
45

17.3
2' F.S.
F
17
3 H.U.
F
61
18.0
3' O.N.
M
60
4' K.I.
F
39
32.7
4' Y.H.
F
57
5' K.I.

5' N.M.
F
41
relapse
F
43
33.4

6' H.K.
F
33
25.3
6' S.H.
F
28
7' U.
F
40
32.6
7' T.S.
F
50

8I.Y.

F

23

32.2

8' M.C.

F

61

9A.N.

F

25

32.1

9' K.I.

F

39

10K.H.

M

29

24.9

10' A.M.

F

33

11 K.U.

F

28

27.6

11' Y.M.

F

50

12T.U.

M

45

25.2

12' I.H.

F

23

13 K.E.

F

17

31.6

13' I.S.

F

21

14T.K.

F

40

28.8

14' I.M.

F

18

15S.H.

F

25

22.8

15' S.K.

F

50

Mean $\pm$ SD 27.39 $\pm$ 5.23 26.71 $\pm$ 6.22

p > 0.05.

Japan the complication of splenomegaly is only 0.8% now. In addition, I scarcely see the word 'splenomegaly' used in connection with hyperthyroidism in recent textbooks on endocrinology. Is it better to think that the effect of RES is rare in Graves' disease? On the other hand, as the mechanism of complication with hyperthyroidism and idiopathic thrombocytopenic purpura is thought to be due to autoimmunity, it was omitted in this letter.

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Table II. Platelet counts during hyper-, hypo- and euthyroidism

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