but they may be ordered and considered by the specialist in this field.—I am, etc.,

C. J. MULDOON

Hull Royal Infirmary,
Hull, Yorks

Personality and Rheumatoid Arthritis

SIR,—I was interested to read Dr. O. W. Hill's letter (5 June, p. 588) regarding my paper on rheumatoid arthritis and personality (8 May, p. 297). It extended, it seemed to me, discussion of the implications of the paper more or less in the manner I had pursued it prior to the ever necessary pre-publication pruning. It emphasized that musculo-skeletal abnormality is likely to affect personality because the two are so inescapably related.

Dr. Hill suggests that a subnormal index of neuroticism (in rheumatoid arthritics) may mean that these are patients who "suppress their awareness of emotional responsiveness," and within this personality framework to be "unable to express hostile or other socially deprecated feelings". This interpretation is of course quite congruent with the contained hostility hypothesis alluded to and to some extent confirmed in my study. However, my paper appeared to categorize such personality reaction in rheumatoid arthritics as post-morbid and not pre-morbid.

It is my hope that the methodology used may be of help in the thorny problem of separating post- from pre-morbid personality features in other "psychosomatic" illnesses, though I would repeat as I did in my paper that neurotic factors can significantly affect the course of an individual's rheumatoid arthritic illness. Put in perhaps crude terminology the disease process can impair the individual's ability "to stand up and fight." This is not surprising when one considers the nature of the disability.—I am, etc.,

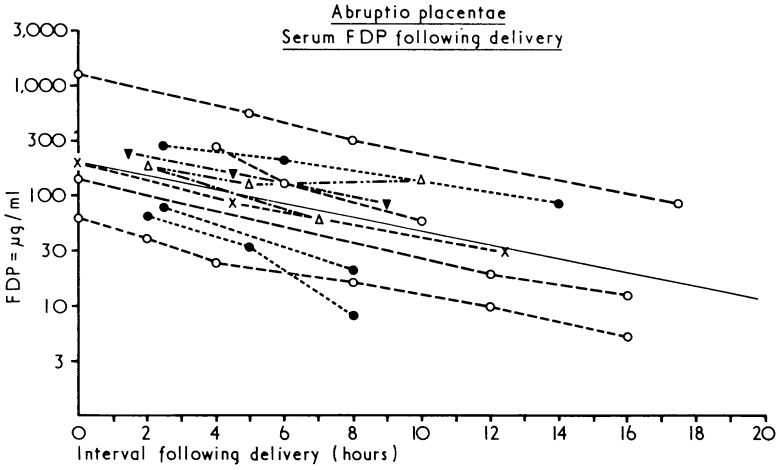
DERMOT J. WARD

St. Loman's Hospital,
Ballyowen,
Palmerstown, Co. Dublin

Postpartum Coagulation Failure

SIR,—Mr. L. Courtney rightly emphasizes the importance of uterine atony in the genesis of postpartum haemorrhage associated with coagulation failure (15 May, p. 403). Coagulation failure may be considered to be a generalized defect determining a tendency to haemorrhage, whereas the local factor in the form of uterine atony (or occasionally genital tract lacerations) determines the occurrence of postpartum haemorrhage. This is because haemostasis from the placental site after delivery is dependent primarily on uterine retraction and not on coagulation of blood. This concept is supported by the observation that 70% of women with hypofibrinogenaemia, those suffering from thrombocytopenic purpura, or those undergoing treatment with anti-coagulants, do not suffer from postpartum haemorrhage unless they develop uterine atony.[1]

In these cases uterine atony is probably not caused by interstitial haemorrhage in the myometrium (Courvelaire uterus), but by haematological changes associated with the development of coagulation defect. It is known that atonic postpartum haemorrhage following abruptio placentae is associated with the presence of high concentrations of fibrin degradation products (F.D.P.) in the serum, particularly the high molecular weight early products of fibrin proteolysis.[1] F.D.P. can inhibit uterine muscle contractility in vitro and be the common link between coagulation failure and atonic postpartum haemorrhage. This inhibitory effect on myometrial contractility is mainly a direct one as F.D.P. can alter muscle membrane activity by altering ionic transfer across the cell membrane.[2] This direct effect is augmented by the potentiating effect of F.D.P. on plasma kinins as F.D.P. inhibit the action of kininase. For example, F.D.P. inhibit the action of kininase on the C-terminal arginine of bradykinin.[3] Bradykinin is known to inhibit the contractility of the pregnant uterus in vitro.[4]



Fibrin degradation products are produced in the circulation as a result of reactive fibrinolysis secondary to disseminated intravascular coagulation. Following a severe degree of premature separation of the placenta, the uterus becomes a site of fibrinolytic activity and releases F.D.P. into the venous drainage of the organ.[5] Thus the concentration of F.D.P. in the uterine circulation, which determines the onset of uterine atony, may be higher than that in the systemic circulation. The figure shows the serum levels of F.D.P. measured by the hamagglutination inhibition method, following delivery in 11 cases of severe abruptio placentae. As can be seen, the serum F.D.P. levels fall fairly rapidly, elimination occurring partly in the urine.[6] Assuming an exponential rate of "decay," the "half life" following delivery appears to be remarkably constant (4·5 hours).

These considerations are important to the management of postpartum coagulation failure. If this is associated with well retracted uterus, postpartum haemorrhage does not occur. If haemorrhage does occur, the treatment should be directed against not only the generalized coagulation defect, but also the local factor in the form of uterine atony (or genital tract laceration). Administration of fibrinolytic inhibitor drugs currently available inhibits further production of F.D.P. but has no effect on F.D.P. already formed. However, following delivery if excessive fibrinolytic activity in the blood is demonstrated by laboratory tests, this implies continued production of F.D.P. (and probably also of plasma kinins) and may indicate the need to administer fibrinolytic inhibitor drugs. The natural clearance mechanism of F.D.P. must be fairly effective as suggested by the relatively short "half life."

The treatment of uterine atony thus appears to be of prime importance, as has been suggested by Mr. Courtney. As uterine atony is probably caused by the direct action of F.D.P. and plasma kinins on myometrial contractility, administration of oxytocic drugs may not be wholly effective in reversing these effects. Mechanical stimulation of the uterus by external massage appears to be the method of choice for the treatment of atonic postpartum haemorrhage. In spite of the encouraging results obtained by Mr. Courtney following uterovaginal packing, it remains possible that the observed beneficial effects could have been due to manual stimulation of the uterus simultaneously instituted. Indeed, could uterine packing have contributed to the "tendency of uterine relaxation" observed in his cases? Another effective method of treatment of uterine atony is the administration of a hot intrauterine douche (116-118°F). This probably acts by inactivating plasma kinins and neutralizing the inhibitory effect of F.D.P. on myometrial contractility.—I am, etc.,

H. K. BASU

Liverpool Maternity Hospital,
Liverpool

1 Basu, H. K., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1969, **76**, 481.
2 Malofiejew, M., Kostrzewska, A., and Buluk, K., *Experientia*, 1971, **27**. 139.
3 Hamberg, U., *Proceedings of the Royal Society, Series B*, 1969, **173**, 393.
4 Landesman, R., Campbell, W. L., and Wilson, K. H., *Obstetrics and Gynecology*, 1963, **22**, 102.
5 Basu, H. K., and Jeffcoate, Sir N., *American Journal of Obstetrics and Gynecology*, 1970, **107**, 1188.
6 Herschlein, H. J., and Steichele, D. F., *Thrombosis et Diathesis Haemorrhagica*, 1968, **19**, 248.

Whose Purpura?

SIR,—May I join Dr. P. W. M. Copeman (5 June, p. 588) in his complaint over the use of the eponym "Henoch-Schönlein" for a certain type of purpura? It is a pity, perhaps, that an eponym has to be used at all, but, as Gairdner pointed out,[1] there is no satisfactory name for the condition and he used "Schönlein-Henoch," which is certainly more accurate than the other way around on historical grounds. Gairdner thought that