can dwell in a man's mind for nine years is surely evidence of mental sickness, the only reasonable and merciful verdict on him and his like. We have been forced to grow used to such things of late and would wish only that they were yet further off, for they have become very much closer than the four centuries that separate us from Servetus and Calvin.

In our issue of January 23, 1954, p. 209, we reviewed Professor O'Malley's *Michael Servetus, A Translation of His Geographical, Medical, and Astrological Writings* (1953). This quatercentenary year has elicited several other works on Servetus. Important among them is that by Professor Fulton, who provides, in most conveniently arranged form, a bibliographical survey of everything that Servetus wrote.* The book is valuable for its critical review of the history of the discovery of the pulmonary circulation. With this discovery Servetus must certainly be credited, pending evidence to the contrary and despite the now well-known reference in a thirteenth-century Arabic writer. The whole subject, proverbially confused, is admirably clarified by Professor Fulton. Perhaps the best way to give an idea of his conclusions is to set out in chronological order the events with which he is concerned.

- 1536(?)–1538(?) Servetus anatomical demonstrator to Guenther in Paris, perhaps with Vesalius.
- 1541 Guenther couples the names of Servetus and Vesalius as his two anatomical assistants.
- 1543 Vesalius's *Fabrica*, published at Basel, does not mention Servetus or pulmonary circulation.
- 1546 Servetus describes pulmonary circulation in an MS.
- 1553 Servetus publishes *Christianismi restitutio* at Lyons. It contains a description of the pulmonary circulation.
- 1555 Servetus burnt at Geneva.
- 1556 The Spaniard Juan Valverde, pupil of Colombo, in a Spanish work printed in Rome, plagiarized Vesalius but describes pulmonary circulation based (?) on Servetus. He does not cite him.
- 1559 The Italian Realdo Colombo in a book published at Venice shortly after his death, and written some years earlier, describes pulmonary circulation based (?) on Servetus. He does not cite him.
- 1616 Harvey's MS. lecture notes contain first mention of circulation as a whole but do not cite Servetus.
- 1628 Harvey's *De motu cordis*, published at Frankfurt, does not cite Servetus.
- 1694 Wotton's *Reflexions on Ancient and Modern Learning*, published in London, contains first public English citation of Servetus and the pulmonary circulation.
- 1706 The philosopher Leibnitz mentions the Servetus discovery in a letter.
- 1723 *Christianismi restitutio* reprinted, in large part, in London but burnt by order of the bishop. Only proof sheets survive.
- 1790 Near-facsimile reprinted at Nuremberg.

Michael Servetus was a theological reformer, scholar, geographer, astrologist, lawyer, mathematician, and physician, and he is usually regarded as the spiritual founder of the Unitarian movement. It cannot be denied that he was violent and fanatical, abusive and intemperate in debate. He was conspicuous for these qualities even in an age in which they were common literary forms. It also cannot be denied that he was a martyr for what he believed to be true. But what he believed, both as to the working of the body in the circulation and as to the nature of the soul, depend, in their ultimate analysis, on the interpretation of a very small number of words, and particularly on the Greek word *pneuma*. That word does, in fact, mean quite different things in the original texts on which he dwelt, and varies also according to the state of physical and physiological knowledge of the time. It is thus not very far from true to say that Servetus died for a single word, and that, for a single word, Calvin took a human life. And it was a word which neither understood or could understand aright.

CHARLES SINGER.

**Michael Servetus Humanist and Martyr*. By John F. Fulton. Bibliography of works and census of known copies by Madeline E. Stanton. (Pp. 98; illustrated. \$8.50.) New York: Herbert Reichner. 1953.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

The Integration of Medicine

SIR,—Sir Harold Himsworth's thoughtful Linacre Lecture (*Journal*, July 23, p. 217) was concerned mainly with diagnosis and less with therapy. He sees British medicine as queasy to-day and likely to be sick to-morrow unless radical changes are made in its organization. There is a growing lack of integration between medicine and its fundamental sciences which has arisen because too few clinicians have been adequately educated in a basic science and too few scientists have received a medical training. The clinicians see their problems but haven't acquired the techniques which would solve them, while the scientists have the skill but lack an intimate acquaintance with disease. No one is so well placed as Sir Harold to analyse these defects in our system, and we must be grateful for his brilliant analysis.

The remedies are, I believe, simple, and only money is needed to apply them. The money has to be invested in men and not in research programmes. The first is the provision of "fellowships" to train young clinicians in the basic sciences. A three-year course with a Ph.D. at the end of it for 20 men annually will require 60 "fellowships" remunerated at £1,500 a year. Six weeks ago I was taken round the Massachusetts Institute of Technology by a young paediatrician who had just completed such a three-year course and was about to return to his work at the Boston Children's Hospital. He had devised an improved method for the production of the thyrotropic hormone. There are many like him in the States, but has he one British counterpart?

The re-entry of such young men into clinical work must be guaranteed. (We have already lost several good young men to the United States and Canada. This is a harsh comment on our present system: medical brains in this country are not so numerous that we can afford to export them.) This will require the provision of associate professorships in clinical research, salaried, let us say, at £2,000 a year. Such associates will be attached to professorial or specialist units, and, in order that they can maintain a clinical competence and a continued contact with sick subjects, should be in charge of the ward service for three months each year. The professor, who has to be master of four trades, would then have a little leisure for thought and research.

Such schemes would cost £500,000 a year; no more than a trivial investment in preventive and therapeutic medicine. The National Health Service has made a good beginning, but it has not yet begun to accept its responsibility for the advancement of medical science. It should not be difficult to persuade Mr. Macleod to steal Sir David Eccles's thunder.—I am, etc.,

Derby.

DOUGLAS HUBBLE.

Anaemia in the Tropics

SIR,—The *Journal* of May 28 has just arrived here, and we have read with great interest Professor A. W. Woodruff's Goulstonian Lecture (p. 1297) on the "Natural History of Anaemia Associated with Protein Malnutrition."

During the past four months we have been investigating the anaemias on the tea estates in the Brahmaputra Valley of Assam. The climate of this region is very hot and damp during the months of April to October and very cool and dry during the three or four months of winter. The patients we investigated were all workers on the tea estates and an extremely uniform group culturally, economically, and

socially. Through the courtesy of the Ross Institute and the Jorehaut Tea Company we were able to collect anaemic patients with Hb below 6 g.% and some below 2 g.% and retain them in hospital for periods of three months or more. During this period they were under our complete control, and full weekly blood studies and serial sternal punctures were done, the results of which will be published later.

The anaemia present in this valley during the hot, damp summer is similar to that which we have investigated in Portuguese East Africa and the coastal regions of Kenya, and is very different to that described by Woodruff in West Africa. These differences pose interesting problems of considerable importance.

None of the anaemias which we have seen here had any liver involvement and very few had enlarged spleens. The patients were men and women, both pregnant and lactating as well as non-pregnant, between the ages of 16 and 35 years, straightforward uncomplicated anaemias of a very severe type such as have been known in this region for many years. They were collected from the tea gardens while still at work. With the exception of one case in 55 they were all microcytic hypochromic iron deficient with M.C.V. between $45 \mu^3$ and $80 \mu^3$ with M.C.H. and M.C.H.C. between 12 $\gamma\gamma$ and 20 $\gamma\gamma$ and 18% to 22% respectively. Their marrows all had a typical erythro-normoblastic picture, sometimes with giant stab cells; a few cases had intermediate megaloblasts. Their total serum proteins were within normal limits, but frequently the albumin-globulin ratio was reversed, the albumin fraction being at the lower limit of normality or below. Gastric acidity after histamine was normal in most cases, as was the indirect van den Bergh reaction. The majority of these anaemias responded to oral iron in doses of 15 to 20 or 30 gr. (1 to 1.3 to 2 g.) daily given in the form of ferrous sulphate for 5 to 10 weeks. They did not respond to lower doses given for shorter periods. Their response to intravenous iron was no better than to oral. Nor was there any difference in the severity of the anaemia, or response to treatment in men, non-pregnant, pregnant, and lactating women. The birth of the child sometimes resulted in a somewhat more rapid response and sometimes not, indicating that pregnancy is not the only factor concerned in this anaemia. During the course of treatment the M.C.V. in all cases rose from the original low level to between $110 \mu^3$ and $150 \mu^3$ with reticulocytes never above 5%. This rise in the M.C.V. during the course of treatment is of considerable interest, since it indicates that macrocytosis is not an index of megaloblastosis and that all the microcytic hypochromic anaemias pass through this macrocytic stage during treatment—hence the importance of sternal puncture for their proper diagnosis and treatment.

It will be seen that these anaemias differ from those investigated by Professor Woodruff in West Africa in that they had no enlarged liver and responded to iron treatment, as was to be expected from their low M.C.H. and M.C.H.C.

Although we have done no critical survey, the diets of these tea-garden workers do not appear to be faulty; in fact they are a good deal better than that of the Macedonian peasantry before the war. Nor would there seem to be any deficiency of iron in the diet. We are therefore doubtful whether nutrition is an important factor in their genesis and maintenance. This anaemia is extremely common in all the hot, damp regions of tropical Africa and India, and its aetiology has not been precisely established. We agree with Professor Woodruff that intestinal parasites are not likely to be an important factor, and malaria is no longer a menace on the majority of the tea estates.

The incidence of these hypochromic iron deficient anaemias is decidedly lower and response to iron treatment more rapid in the cooler parts of the year, and in places of higher altitude. For example, on the hot, damp coast of Kenya this is the predominant anaemia, but in the cooler highlands it gives place to the megaloblastic hyperchromic type, the incidence of which has also been shown to be seasonal.¹ The common denominator in all the areas

where these iron-deficient anaemias are so abundant is great heat and humidity; variations in diet and the incidence of intestinal parasites do not show any close correlation. Although, of course, more than one factor may be involved, it seems that iron loss through excessive perspiration may be the most important. Work is proceeding on this question and will be published separately. Nitrogen loss in the perspiration has also been shown to be excessive under such conditions, and this may be a factor in the protein situation.

As a result of our work we have been able to divide these hypochromic microcytic iron deficient anaemias of the damp, hot Tropics into the following groups: (1) Those with erythro-normoblastic marrows and normal albumin: this group responds adequately to iron unless they develop giant stab cells or their albumin falls during the course of treatment. (2) Erythro-normoblastic marrows but low serum albumin: this group responds partially to oral iron but needs albumin supplements in the form of skimmed milk to bring them to their normal blood values. (3) Giant stab cells in their marrows but normal albumin: this group responds partially to oral iron but needs 25 mg. to 40 mg. of folic acid for complete response. (4) Those with giant stab cells and low albumin go up partially on iron but need both proteins and folic acid to produce normal blood picture; they do not go up with albumin alone, folic acid alone, or iron alone. (5) Those with megaloblastic marrows which do not go up with iron or B₁₂ and folic acid but do so with folic acid and iron. (6) Cases with erythro-normoblastic marrows which respond partially to iron, then develop the giant stab cells and/or low albumin and then require the treatment outlined above.

We are aware that these giant stab cells occur in other conditions than anaemia, but their presence in the anaemias that fail to respond to iron or proteins, their appearance in those that are having improper treatment, and their disappearance when folic acid is given all seem to indicate that they have some significance in anaemic conditions.

These microcytic iron deficiency anaemias of the Tropics and subtropics are far more complicated and more prevalent than are the hyperchromic megaloblastic, and there appears to be no one single factor either in their aetiology or treatment that can be regarded as paramount.—We are, etc.,

HENRY FOY.
ATHENA KONDI.
B. SARMA.

Assam, India.

REFERENCES

- 1 Foy, H., et al., *Lancet*, 1955. In press.
- 2 Patwardhan, V. N., *Nutrition in India*, 1952. Bombay.

Resuscitation in Asphyxia Neonatorum

SIR,—Dr. Bryan M. Hibbard (*Journal*, July 16, p. 183)¹ describes the case of an infant who, after being resuscitated from severe post-natal asphyxia, subsequently had symptoms of severe injury to the brain, and at 12 months evidence of gross mental retardation with epilepsy. Dr. Hibbard asks how long an infant can sustain anoxia and subsequently show normal development; he refers to the case reported by Holmes and Payne in the *British Medical Journal*¹ in which the infant recovered following prolonged endotracheal oxygen, and questions whether such prolonged efforts at resuscitation are justified.

I write to inform you that the child whose case was reported by Dr. Holmes and myself has subsequently been followed up by Dr. D. G. Leys at Farnborough Hospital, and at 18 months this child's progress is in every respect normal. This case, then, is evidence of the fact that the newborn child may sustain a period of 20 minutes' anoxia without apparent damage to the brain. It is of course possible that this child may later show some evidence of having suffered injury, but normal progress to the age of 18 months would ordinarily be taken as strong indication to the contrary.—I am, etc.,

Maidstone, Kent.

J. A. PAYNE.

REFERENCE

- 1 Holmes, J. A., and Payne, J. A., *British Medical Journal*, 1955, 1, 1071.