Correspondence : Briewerubriek

The views expressed in the Correspondence published in the SAMJ *are not necessarily those of the Medical Association of South Africa.*—Editor.

Die menings gelug in die Briewerubriek van die SAMT *is nie noodwendig dié van die Mediese Vereniging van Suid-Afrika nie.*—Redakteur.

DELAYED RESPIRATORY DEPRESSION WITH FENTANYL

To the Editor: With regard to the letter on the above-mentioned subject by Cajee and Buchanan,[1] I should like to make the following comments:

While the articles by Adams and Pybus[2] and Becker *et al.*[3] are valuable and raise doubts about fentanyl's being a 'short-acting' drug, perhaps the most telling evidence against these doubts is contained in an article by Morgan *et al.*,[4] which reported a study of 500 cases of neurolept anaesthesia for major surgery. The authors found that 'respiratory depression after operation did not recur in any patient'. Coming as it does from an institution noted for its impeccable application of scientific observation, this statement must weigh heavily in any argument on neuroleptic anaesthesia using fentanyl and 'late' respiratory depression.

It is notable that Morgan *et al.*[4] used a 'pure' neuroleptic technique, i.e. droperidol and fentanyl only as induction agents, whereas Adams and Pybus[2] used other induction agents to supplement the droperidol-fentanyl combination. Perhaps there is a connection between the use of additional induction agents and recurrent respiratory depression.

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1. Cajee, R. and Buchanan, N. (1978): S. Afr. med. J., **54**, 810.
2. Adams, A. P. and Pybus, D. A. (1978): Brit. med. J., **1**, 278.
3. Becker, L. D., Paulsen, B. A., Miller, E. R. *et al.* (1976): Anaesthesiology, **44**, 291.
4. Morgan, M., Lumley, G. and Gillies, I. D. S. (1974): Brit. J. Anaesth., **46**, 288.

PLACENTAL SIZE AT BIRTH

To the Editor: It was with great interest that I read the article by Woods *et al.*[1] in the *SAMJ* of 4 November 1978. I fully agree that the basic premises appeared to be correct, but there is one aspect of the methods and conclusions which may require further investigation before final acceptance is possible. It is mentioned (on page 778 of the article) that 'placentas were stored at 4°C and examined within 24 hours of delivery', that 'the umbilical cord was cut flush with the chorionic surface' and that 'the trimmed placenta was then weighed'.

On page 779, it was stated that 'The thickness reflects the amount of parenchyma, and the volume of retained intervillous maternal blood. Most of the latter had leaked out by the time the placentas were assessed, which suggests that the increase in thickness during late pregnancy is due to accumulation of parenchyma.'

I wish to point out that, for the following reasons, there may have been an error in the method and conclusions. Obstetricians know that in the case of some individuals, or in some centres, the placental end of the cord may either be left clamped until quite some time after the confinement has been completed, or the cord may be unclamped, or even cut to allow as much blood as possible of the placental-fetal circulation to drain from the placenta immediately after the cord has been clamped on the fetal side. The latter method allows for a substantial reduction in the intravillous pressure, thus reducing the chances of transplacental fetal haemorrhage. At the same time, the substantial fetal blood drainage from the placenta reduces the placental size and therefore enhances the chances of an easy delivery of the smaller placenta by the Brandt-Andrews method of placental delivery, which is commonly used by most obstetricians today.

Apropos of the above methods, we also know that during accidental prolapse of the cord, spasm of the cord vessels very frequently results in cessation of the fetal circulation owing to temperature, humidity, handling and other factors, without compression playing any part. Spasm of the cord is also one of the methods whereby the cord circulation stops and prevents fetal bleeding after birth in many animal species.

It would therefore appear that without a standardized method of delivery of the placenta, a theoretically greater or lesser amount of fetal blood might have remained in the intravillous system within the placenta, thus influencing the final size and weight after the membranes had been trimmed, the cord had been cut flush after storage at 4°C for 24 hours after delivery, and adherent blood clots had been washed free.

With regard to the maternal intervillous space blood, this problem may also occur, but to a lesser extent. Maximal drainage of maternal blood from the intervillous space may also be influenced by whether the cord remains clamped, thereby retaining a large intravillous volume, and thus expressing maternal blood from the intervillous space during shearing off of the placenta through the decidual layer, as well as during the further method of delivery of the placenta. In addition, the marginal sinuses may not necessarily be of equal size around the edges of all the placentas, thus resulting in differences in drainage through this source. In addition, the placentas may not be stored in the refrigerator in exactly the same manner, so that some may be squeezed side-to-side while others may be lying flat, or they may even be piled up one on top of the other.

These factors could all influence variability in both the intravillous volume and the intervillous volumes in individual placentas, and thus also their weight and size.

One would be very much happier if a study whereby all these variables were standardized were carried out.

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1. Woods, D. L., Heese, H. de V., Davey, D. A. *et al.* (1978): S. Afr. med. J., **54**, 776.

WITCHDOCTOR OR WHICH DOCTOR?

To the Editor: I think that Professor T. Dunston,[1] in his rather scathing comment on Dr Ferguson's[2] letter, is confusing the practice of medicine with the science of medicine. To cite the disagreement between radiotherapists, chemotherapists and surgeons on the matter of the treatment of malignant disease as an example of the lack of scientific principles in medicine is to ignore the mass of literature produced every day on the very subject of disagreement in understanding of disease and the treatment thereof. It simply denotes viable and vigorous thinking together with a study of the science and application of its principles.

Since it was written by the Dean of a medical faculty, this letter will no doubt have raised eyebrows, possibly indulgently, since he can hardly be taken seriously.

By his own admission he is cynical, and cynicism knows the price of everything, but the value of nothing.

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1. Dunston, T. (1978): S. Afr. med. J., **54**, 1043.
2. Ferguson, D. L. (1978): *Ibid.*, **54**, 810.

DIPHENYLHYDANTOIN DURING PREGNANCY

To the Editor: With reference to the 'News and Comment' item 'Diphenylhydantoin during pregnancy',[1] I should like to comment on the possible underlying mechanism of the anticonvulsant-induced fetal malformations, namely folate deficiency.

That repeated administration of certain drugs may result in induction of the hepatic microsomal drug-metabolizing enzymes has been well documented. The activity of these enzymes may be affected by nutritional status, and by the same token, the prolonged administration of drugs may, because of increased nutrient requirements, result in the manifestation of certain nutrient deficiencies and toxic effects.[2]

In a study of the nutritional status of patients on long-term anticonvulsant, phenothiazine or tricyclic drug therapy, the serum and erythrocyte folate concentrations in patients on long-term anticonvulsant or phenothiazine therapy were significantly lower than those of control subjects on the same diet.[3,4] Biochemical folate deficiency was further substantiated by increases in urinary formiminoglutamic acid excretion after histidine loading. A significant increase in the urinary excretion of D-glucaric acid was indicative of hepatic microsomal enzyme induction. The study revealed that the folate deficiency was a function of the duration of drug administration, and was not determined solely by the nature of the drug. Patients receiving anticonvulsant, phenothiazine or tricyclic drugs for a period of 2 - 5 years showed marked microsomal enzyme induction, and evidence of folate deficiency became manifest after about 5 years of drug administration. However, as the period of treatment was extended to 10 years, folate deficiency increased, but enzyme induction decreased. It is suggested that the decrease in enzyme induction may be the result of the increasing folate deficiency. This may explain the sudden appearance of symptoms of drug overdosage and toxicity in patients stabilized for several years on a particular dose of an anticonvulsant drug. In addition, any decrease in enzyme induction may be responsible for elevated blood levels of anticonvulsant drugs, and since diphenylhydantoin is transported across the placenta, the fetus may be exposed to toxic drug levels.

The administration of phenobarbitone or diphenylhydantoin to rats for 12 weeks resulted in marked folate deficiency and absence of microsomal enzyme induction.[3] Furthermore, folate deficiency is associated with a marked reduction in the activity of the hepatic microsomal drug-metabolizing enzymes (D. Labadarios — in preparation). These effects can be ameliorated by the administration of folic acid supplements.

In similar experiments[3] on pregnant rats dosed with phenobarbitone plus diphenylhydantoin for 12 weeks before mating and during gestation, the mean fetal and litter weights of the newborn of rats maintained on a folate-deficient diet were markedly reduced when compared with control animals on the same diet. Furthermore, these neonates exhibited marked deformities, including reduced bone mass and ossification, spinal malrotation, hydrocephaly, head and abdominal haemorrhages, hydronephrosis and hydro-ureter. The impairment in reproduction and the appearance of neonatal abnormalities progressively decreased from 'drug-treated folate-deficient' animals to 'control folate-deficient' to 'drug-treated, folate supplemented' animals with 'control folate-supplemented' rats as normal controls. These findings confirm previous observations of the effects of folate deficiency on skeletal development and organ abnormalities, and substantiate the various reports of congenital malformations seen in the offspring of women on prolonged anticonvulsant therapy.[5,6]

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1. News and Comment (1978): S. Afr. med. J., **54**, 726.
2. Maxwell, J. D., Hunter, J., Stewart, D. A. *et al.* (1972): Brit. med. J., **1**, 297.
3. Labadarios, D. (1975): 'Studies on the effects of drugs on nutritional status,' Ph.D. thesis, University of Surrey, Guildford, Surrey, England.
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6. Speidel, B. D. and Meadow, S. R. (1972): Lancet, **2**, 839.

ULTRASONIC MAMMOGRAPHY

To the Editor: In reply to Dr Smit's[1] letter about ultrasonic mammography screening, may I point out that at present this is not possible?

Highly sophisticated Greyscale machines can be used to differentiate cystic from solid breast masses, if these are palpable. For general breast ultrasonic mammography, however, special apparatus and the use of a water bath and the prone position are necessary. This apparatus is extremely expensive, and is not yet in general use. It would therefore appear that at present ultrasonic examination can only be a complementary procedure in our investigative armamentarium.

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1. Smit, B. J. (1978): S. Afr. med. J., **54**, 846.

MUST PHARMACISTS ONLY COUNT PILLS?

To the Editor: Mrs Lois Kay, ex-Chairman of the Natal Coastal Branch of the South African Retail Chemists' Association, is reported[1] to have said, in her annual report, that 'those pharmacists who have to contend with dispensing doctors have little to encourage them to continue with their profession'.

Recently the Chairman of the Federal Council of the Medical Association of South Africa[2] asked: '. . . op watter wyse kan ons dit regverdig dat die bevordering van die apteker se belange gepaard moet gaan met die aftakeling van geneesheerdienste op die platteland?'

The contemporary discussions as to who should and who should not dispense are not confined to our country. Pharmacists are trying very hard to discard their shopkeeper image and to find a niche for themselves in the health team. Boylan[3] proposes the following: 'Advising patients on self medication is the pharmacist's major contribution to health education.' Her view is echoed by Freed,[4] who says: 'They have specialized training, they are professionals and they must be regarded as consultants.' He also feels that 'In his capacity as a consultant he should be entitled to a fee'.

From the UK[5] comes the following statement: 'There is a trend within pharmacy towards a consensus that the future lies with, for want of a better term, clinical pharmacy. While the term may be open to various interpretations, it seems clearly to embrace the general idea that pharmacists should have a greater involvement with the care of the patient.' Does this mean that the pharmacist should take automated blood pressure and pulse readings?

It is clear that consumerism is the culprit, and this is illustrated by the limited survey conducted by Boylan. Her findings were that 'Most queries resulted in a purchase'. Only 15 - 18% of people who presented with skin disorders or gastrointestinal symptoms were given advice, and were not recommended to make a purchase.

Thus, there is a conflict between two parties who should be working in harmony. To ameliorate the situation, the first requirement is that both parties analyse the position unemotionally and avoid *faux pas* at all costs. No one will deny that the knowledge of the truly conversant pharmacist could and should be better utilized, but this should never be done at the expense of other people, especially the patients. We must realize that both professions are independent but at the same time interdependent, and that maximum benefits will only accrue when both parties are serious about patient care.

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1. Kay, L. (1978): *Retail Chemist*, **26**, 9.
2. De Klerk, J. N. (1978): S. Afr. med. J., **54**, 955.
3. Boylan, L. J. (1978): *The Pharmaceutical Journal*, p. 328.
4. Freed, E. D. (1978): S. Afr. med. J., **54**, 999.
5. *The Pharmaceutical Journal*, **221**, 313 (1978).

GENEESKUNDE-ONDERRIG

Aan die Redakteur: Gedurende die onlangse M.B. Ch.B.-eindeksamens het ek die voorreg gehad om by twee mediese skole as eksaminator op te tree — by die een as interne en by die ander as eksterne eksaminator. Hierdie betrokkenheid by die eksamens het my opnuut bewus gemaak van die volgende twee probleme.

Ten eerste tref dit 'n mens dat daar van mense verwag word om mondelinge eksamens af te neem sonder dat hulle enige opleiding in die afneem van eksamens gehad het. Gewoonlik word 'n junior en 'n senior eksaminator saamgegroepeer, en leer die juniors mettertyd van die senior eksaminatore hoe om te eksamineer. Ongelukkig is alle senior eksaminatore nie noodwendig goeie eksaminatore nie, en dit gebeur soms dat 'n eksaminator sy nuutste kennis aan sy mede-eksaminator wil toon deur die vrae wat hy aan die student stel. Ook vind 'n mens dat 'n eksaminator in diepte op 'n onderwerp ingaan sodra hy agterkom dat die student onseker omtrent daardie spesifieke onderwerp is. Elke eksaminator tree maar op soos hy dink hy moet, of soos hy 'n vorige mede-eksaminator sien optree het. Professor F. P. Retief[1] het in die *SAMT* van 25 November 1978 na hierdie gebrek by eksaminatore, om die student reg te beoordeel, verwys. Myns insiens is dit nou tyd dat universiteite op 'n georganiseerde wyse hul personeel oplei om as eksaminatore op te tree, veral wat die mondelinge eksamens betref.

Ten tweede het 'n mens weer besef hoe geweldig belangrik 'n goeie teoretiese kennis is. Die student wat sy werk ken, al het hy nog nie veel praktiese ondervinding van sy vak opgedoen nie, is een wat (*i*) met selfvertroue sy mediese beroep kan aanpak, wetende dat hy 'n grondige kennis het; (*ii*) oorspronklik kan dink, ook betreffende moontlike navorsing wat hy later mag doen; en (*iii*) daarin slaag om die praktiese deel van sy beroep gou onder die knie te kry omdat hy 'n deeglike teoretiese kennis het. Myns insiens is die handhawing van 'n hoë akademiese standaard, ook veral wat die teorie betref, uiters belangrik. Met die nuwe leerplan wat geleidelik in al die mediese skole in ons land ingevoer word, nl. dié van 5 jaar studie en 2 jaar sg. internskap (alhoewel die 1ste jaar van die internskap ook nog met kliniese demonstrasies en besprekings gepaard sal gaan), is daar net nie genoeg tyd om die teoretiese kennis op te doen wat die student by die einde van sy opleiding behoort te hê nie. Dosente met ondervinding sal beaam dat die gemiddelde student 'n metamorfose vanaf die einde van sy 5de na die einde van sy 6de jaar ondergaan, omdat sy kennis in die loop van hierdie jaar geweldig uitbrei. Volgens die nuwe leerplan moet die student 'n semi-eindeksamen aan die einde van sy 5de jaar aflê, en gedurende die 6de jaar is die stimulus om hom verder teoreties te bekwaam ongelukkig nie meer daar nie. Dit is vir my moeilik verstaanbaar wat die praktiese ondervinding wat hy in sy 6de jaar opdoen (bv. die neem van bloed by pasiënte), en wat hy in sy 7de jaar as intern sal moet herhaal, hom sal baat. Weens die losmaking van die student van sy teoretiese studie in die 6de jaar, sien ek nie hoe ons dieselfde standaard van kennis soos in die verlede sal kan handhaaf nie. Waarom ons 'n stelsel wat in die verlede goed gewerk het — kyk maar net na die geneeshere wat tot dusver in ons land afgestudeer het en die getal wat hoofde van departemente in ander lande geword het — moes verander na 'n stelsel wat herinner aan die opleiding van 'n geneesheer gedurende die begin van die vorige eeu, toe hy hom as leerjonge by 'n praktiserende dokter moes inskryf, kan ek net nie verstaan nie. As ek iets het wat goed werk, hou ek daaraan vas.

Ter wille van ons jongmense wat hulle as geneeshere wil bekwaam, hoop ek dat ek in die duister tas. Dit is jammer dat slegs die tyd dit sal leer.

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1. Retief, F. P. (1978): S. Afr. med. J., **54**, 883.

FRACTURES OF THE TIBIAL SHAFT

To the Editor: Congratulations to Professor Dommisse[1] for advocating the conservative treatment of fractures of the tibial shaft.[1] All experienced surgeons know the folly of plating compound fractures, and the danger of plating closed fractures. Modern electrical and pneumatic tools are great fun to use, but if one feels that one must plate something, then the fibula should be plated.

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1. Dommisse, G. F. (1978): S. Afr. med. J., **54**, 1021.

ABORTUS EN HET FOETALE ALCOHOL SYNDROOM

Aan de Redakteur: In Uw editoriaal 'The fetal alcohol syndrome'[1] stelt U: 'A good case can be made out for termination of pregnancy in alcoholic mothers, ... Meanwhile, there are some unanswered questions, complicated by the fact that the fetal alcohol syndrome is probably only the full expression of a toxic effect that can range from very minor physical and mental defects to serious handicap.'

Waarom U blijkbaar de VSA suggestie in het artikel van Hayden en Nelson[2] overneemt als 'A good case . . .', ontgaat mij volledig. De argumenten, van medische implikaties en de daaruit voortvloeiende sociale last, doen denken aan de goede Middeleeuwse tradities van het behandelen van de 'pest' door het bijeendrijven van de pestlijders in hun huizen, om ze daarna in brand te steken. Geneesheren zoals Pasteur, die met veel ijver de pestbacil en zijn mechanismes ter voorkoming van de verspreiding van de ziekten hebben bestudeerd, moeten zich wel omdraaien in hun graf bij het lezen van de door U aangehaalde stellingen.

Het is dus nu niet alleen de zekerheid omtrent foetale afwijkingen, maar het feit dat moeder drinkt, wat reeds als voldoende wordt beschouwd om abortus als 'alternatief' in te roepen. Waarom doet U geen voorstel voor kwashiorkor, mentale instituten, etc. U weet toch dat in Europa heelwat kritiek wordt uitgeoefend op de oorzaken van deze sociale kwaal, alcoholisme, die door bepaalde instanties voor bepaalde bevolkingsgroepen wordt veroorzaakt, of in stand gehouden. Wilt U dan aan de behandeling hiervan abortus nu nog als middel toevoegen? Of was het misschien niet wijzer, juist deze sociale oorzaken van drankzucht te bestrijden met veel moed en volharding in het licht van de soms tragische gevolgen, eerder dan abortus als gemakkelijkheidsoplossing voor te stellen, alsof dit moreel verantwoord zou kunnen zijn.

Als iemand die steeds van Zuid-Afrika is blijven houden, en die de morele standaarden van het Zuid-Afrikaanse volk en zijn leiders hoog aanslaat, doet het me pijn om in het leidinggevend medisch orgaan dergelijke ongefundeerde en mode-getinte uitspraken te zien verschijnen; waarbij de diepe gronden van 'of iemand wel ooit kan beschikken over het leven van iemand anders' zelfs niet meer ter diskussie worden gebracht.

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1. Editoriaal (1978): S. Afr. med. J.,**54**, 571.
2. Hayden, M. R. en Nelson, M. M. (1978): *Ibid.*, **54**, 571.

The statement 'A good case can be made out . . .' does not imply that all mothers who drink may now have an abortion. It refers to continuous heavy drinking during pregnancy. We in the Republic of South Africa have not yet decided to 'beschikken over het leven van iemand anders'! — Ed.

TOXOPLASMOSIS AND CLINDAMYCIN

To the Editor: Symptomatic acquired toxoplasmosis may be difficult to diagnose and treat. An unusual presentation and response to novel therapy prompts us to report this complex case.

A 20-year-old Black Rhodesian youth was admitted on 18 May 1977 from a mission hospital where he had been ill for some days. He had had substernal pain for 3 weeks, and had grunting respiration, peripheral oedema, elevated external jugular pressure, and a pulse rate of 68/min, but there was no radiological evidence of pulmonary oedema. His pupils were of normal size. An ECG revealed a sinus arrhythmia. His temperature was 39°C, and his white cell count was 15 300/μl. The next day his condition had deteriorated. The PR interval was 0,32/s at a rate of 70/min.

Although the patient assisted in a market garden, there was no history of exposure to insecticide, and his serum cholinesterase level was 1,5 U/ml (normal 3,0 - 8,0 U/ml). Later levels were 0,4 U/ml and 0,5 U/ml. A cholinesterase reactivator was given, and treatment with atropine was commenced and continued for 9 - 10 days. On some days he required 9 mg/24 hours to maintain a pulse rate above 90/min. By day 6 his ECG was normal and he had recovered, apart from persistent pyrexia, initially attributed to the atropine treatment.

Toxoplasma myocarditis had been considered in the differential diagnosis, but was dismissed when organophosphorus poisoning was diagnosed. Low-grade fever continued for 8 weeks. The white cell count remained between 15 and 18 x $10^3/\mu$l and the erythrocyte sedimentation rate was consistently 80 - 90 mm/1st h. Blood cultures, aerobic and anaerobic, were persistently negative at 10 days.

Toxoplasmosis was reconsidered when atypical mononuclear cells were found in the peripheral blood. The 'Monospot' test was negative, but the *Toxoplasma* HA test was positive, as was the toxoplasmosis fluorescent antibody test (dye test unavailable). A small supraclavicular node was detected and subjected to biopsy. The architecture of the node was preserved, but the medulla was locally peppered with collections of histiocytes. The appearances were thought to be consistent with a diagnosis of toxoplasmosis, and on review we believe that the histological appearances fulfil the criteria for the diagnosis of acute *Toxoplasma* lymphadenitis.[1]

Treatment was attempted with co-trimoxazole in double dosage, but there was no remission of fever by day 8. At this point one of us (A.E.M.) returned from South Africa, where he had met Dr H. A. Feldman, who had stated that clindamycin was effective against toxoplasmosis in the laboratory animal, but that as the drug had not yet been generally released in the USA, he was unaware of its effectiveness in man.

Our patient was given oral clindamycin 600 mg daily. Defervescence ensued and by day 9 there was complete remission of fever. The drug was continued for 28 days, and when the youth was discharged on 15 August 1977 he was apyrexial and clinically well. On 7 October 1977 his serum cholinesterase level was 1,3 U/ml!

Organophosphorus poisoning is a frequent cause of admission to hospital in Rhodesia.[2] Chronic exposure to organophosphorus compounds was suggested by the low cholinesterase level at 5 months. The patient's dysrhythmia was probably caused by *Toxoplasma* myocarditis; it is interesting that atropine appeared to correct this dysrhythmia.

Clindamycin has been used with some success in the treatment of ocular toxoplasmosis.[3] We should like to see this extended to attempted management of systemic forms of the disease.

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1. Dorfman, R. F. and Remington, J. S. (1973): New Engl. J. Med., **289**, 878.
2. Mackintott, M. E. and Crozier, D. E. (1978): Cent. Afr. J. Med., **24**, 41.
3. Tate, G. W. and Martin, R. G. (1977): Canad. J. Ophthal., **12**, 188.

APUDOMAS AND THE GASTRO-INTESTINAL TRACT

To the Editor: I read with interest your editorial[1] on 'APUDomas and the gastro-intestinal tract', and note that you are promulgating the view that APUD cells appear to arise in the neural crest. I know that this has been widely but uncritically accepted by many, and should like to make the following comment:

Pearse's original proposal that all APUD cells, and therefore gut and pancreatic endocrine cells (which he included in the APUD series), are derived from the neural crest has not been substantiated. There is in fact evidence that one gastro-intestinal cell type, the enterochromaffin cell, does not have such a source;[2,3] the same applies to the major pancreatic endocrine cell types.[4-6] Pearse himself has accepted this evidence,[7,8] and now suggests that all APUD cells are derived from 'neurendocrine-programmed epiblast'. There is no evidence in favour of this either, and it is not experimentally testable at present.

Much of the evidence against a neural crest origin for some gastro-intestinal and pancreatic endocrine cells was analysed in my review,[9] which was published a couple of years ago.

As it is no doubt going to be a struggle to expunge the 'neural crest idea' from people's minds, I hope you will see fit to help by publishing my comment.

A. Andrew

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1. Editorial (1978): S. Afr. med. J., **54**, 996.
2. Andrew, A. (1963): J. Embryol. exp. Morph., **11**, 307.
3. *Idem* (1974): *Ibid.*, **31**, 589.
4. *Idem* (1976): *Ibid.*, **35**, 577.
4. Pictet, R. L., Rall, L. B., Phelps, P. *et al.* (1976): Science **191**, 191.
6. Fontaine, J. L., Le Lievre, C. and Le Douarin, N. M. (1977): Gen. comp. Endocr., **33**, 394.
7. Pearse, A. G. E. (1975): Folia anat. jugoslavica, **4**, 5.
8. *Idem* (1977): Med. Biol., **55**, 115.
9. Andrew, A. (1976): S. Afr. med. J., **50**, 898.

WIDDICOMBE FAIR

To the Editor: In connection with the case report on 'Skin necrosis after warfarin therapy', published on page 609 of the *SAMJ* of 7 October 1978, by M. S. Elliot, E. J. Immelman, P. Jeffery, S. R. Benatar, M. R. Funston, J. A. Smith, P. Jacobs, B. J. Shepstone, A. D. Ferguson and J. H. Louw, we wish to congratulate the authors.

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D. E. Fell
G. Hunter
I. J. Karam
L. M. Niewenhuizen
O. P. Quan
R. Scott
T. U. van der Merwe
W. X. Yarrow
Z. Zat

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Is there a patient in the house? — Ed.

BOUQUETS, NOT BRICK-BATS!

To the Editor: Allow me to pay tribute, through the *SAMJ*, to those doctors who take the trouble to write out their prescriptions carefully and neatly. You have no idea how pleasant it is to be able to see at a glance what is required.

To those doctors I say, 'thank you'; every time I receive one of your scripts you make my day!

F. Eckard

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