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In reply:—We appreciate Dr. Linko's interest in our study and hope that our answers will clarify his inquiries.

1) The screen filtration pressure (SFP) extrapolations were obtained from the straight portion of the Bentley transducer which is linear to 900 torr, not 500 torr.

2) Harp *et al.*[1] demonstrated a profound increase in microaggregate formation, measured by screen-filtration pressure between day 6 and 8 of storage. Thereafter, a slow, persistent rise in SFP was apparent through day 21. We have assumed that three- to six-week-old blood would continue to accumulate microaggregate debris at a similar slow rate.

3) We agree that filtration of erythrocyte concentrates cannot be exactly compared with whole-blood microfiltration. For just that reason, we studied filtration of erythrocyte concentrates since our hospital rarely uses whole blood.

4) Although the standard 170-μm filter is changed after each unit of blood passes through it, we do not recommend this practice. We agree that there is no reason to discard the 170-μm filter after a single use.

5) We concluded that fine-screen filtration was safe, not just because of our own data but because of the studies by Marshall *et al.*[2,3] to which we referred.

6) We agree that the study by Durtschi, *et al.*[4] suggests that filtration through a 40-μm mesh filter offers no significant protection to pulmonary function in man. Their *in vitro* findings are qualitatively similar to ours since only 12 per cent of their microaggregates were filtered. We agree that the more efficient filters need to be studied during massive transfusion just as Durtschi *et al.* have done with the 40-μm mesh filter. Since our studies show that the more efficient filters (Bentley®, Fenwal®, Biotest®) allow excellent flow rates of packed cells infused under pressure, studies of these filters are needed.

7) It might be possible to transfuse buffy-coat free blood as suggested by Högman *et al.*,[5] but preparation of blood with the saline-adenosine-glucose additive is not yet licensed for use in the United States, nor is it available to us through our blood bank.

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The Use of Saline for the Differential Diagnosis of Pain Mechanisms

To the Editor:—The recent Clinical Report by Benzon *et al.*[1] documents again that the conduction of nerve impulses can be blocked by agents other than those usually associated with local or regional anesthesia. While it is true that we have recommended differential neural blockade "to differentiate psychogenic, sympathetic, and somatic sensory-mediated pain," we have never stated that "pain relieved by injection of physiologic saline solution is regarded as psychogenic in origin" categorically, as stated by the authors. We have been extremely careful to state that "relief after the injection of saline suggests a psychogenic basis for the pain," and have indicated clearly that "to allow accurate interpretation of the results of differential blocks, in addition to noting the subjective response of the patient, objective evaluation of sensory (pinprick), motor (mobility), and sympathetic (skin temperature, oscillometry, psychogalvanic response) func-

tion should be undertaken." A diagnosis is established using differential neural blockade *not* by correlating what concentration of local anesthetic has been injected (including zero per cent) with the onset of pain relief, but rather by correlating what modality has been blocked with the onset of pain relief. Just as one could not make a diagnosis of a sympathetic mechanism if a stellate ganglion block *with local anesthetic* does *not* produce a Horner's syndrome, similarly one cannot make a diagnosis of a psychogenic mechanism if the injection of a "placebo" does produce a Horner's syndrome. This concept is absolutely essential if differential neural blockade is to be used *effectively* in the differential diagnosis of pain mechanisms.

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A Further Statement on Automated EEG Processing for Intraoperative Monitoring

To the Editor: — With their article, "Automated EEG Processing for Intraoperative Monitoring," Levy *et al.*[1] have in our opinion, rendered a service to anesthesiologists by sorting out and clarifying the several EEG analytical systems under development at this time. In their article they credit us with having worked on what appear to be two systems of EEG analysis: "multiple differential analysis" and "period amplitude analysis," thus potentially creating the impression that we have developed two different EEG analytical techniques. We wish to point out with a short explanation of our technique, that these are not two analytical systems, but rather, what the authors call "period amplitude analysis" (PAA) is actually a subset of a larger analytical system which they refer to as "multiple differential analysis." In our original publications we preferred to use the expression "derivative analysis" instead of "multiple differential analysis" to refer to the larger technique, and hence will use this former terminology for our explanations presented here.

In 1976 we described our method of derivative analysis and its performance under different conditions of anesthesia.[2] The method used a six-parameter, time domain derivative, wave analyzer based in part upon period analysis.[3,4] For a given single-channel EEG input, this analog analyzer extracts the following six features from the incoming EEG wave:

F0: The number of zero-axis crossings of the original wave, per unit time. This parameter is referred to as basic frequency (Units: hertz).

A0: The average rectified amplitude of the original wave. This parameter is referred to as basic amplitude (Units: volts).

F1: The number of zero-axis crossings of the first derivative of the original wave, per unit time. This parameter is referred to as first derivative frequency (Units: hertz).

A1: The average rectified amplitude of the first derivative of the original wave. This parameter is referred to as first derivative amplitude (Units: volts/s).

F2: The number of zero-axis crossings of the second derivative of the original wave, per unit time. This parameter is referred to as second derivative frequency (Units: hertz).

A2: The average rectified amplitude of the second derivative of the original wave. This parameter is referred to as second derivative amplitude (Units: volts/s^2).

It is the first two parameters (F0, A0) which Levy *et al.* refer to when they speak of "period amplitude analysis" (PAA). Our F0 parameter, corresponds to their zero cross frequency (ZXF), and our A0 parameter corresponds to their mean rectified voltage (MRV). We are more comfortable with the notion that we have developed one system of EEG analysis of which it is often adequate to use only the lower two parameters (F0,A0) for particular applications.

We have in fact spent some time in developing and