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Letter to the editor

Comments on "Nerve conduction as a means of estimating early postmortem interval" by K. J. Straton, A. Busuttil and M. A. Glasby

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Dear Sir,

The authors studied the nerve conduction as a means of estimating the early postmortem interval (pmi).

The main finding is that the chronaxie of the rat sciatic nerve increases in the first 90 min p.m., after which a plateau phase is reached.

Corresponding to the experimental design (duration of stimuli only between 0.05 and 4 μs) the rheobase as well as the chronaxie are *calculated,* not measured values.

Experiments on strength – duration curves of skeletal muscle postmortem (rats, dogs, humans) have shown that much longer durations of stimuli are necessary, for measuring the rheobase, especially with increasing pmi (Joachim 1976; Madea 1989, Madea and Henßge 1991).

For determining the rheobase of skeletal muscle, stimuli of 1000 msec duration are used (Joachim 1976; Joachim and Feldmann 1980; Madea and Henßge 1990b).

In Fig. 7 of the paper, 5 mean values of the chronaxie (at the moment of death, 5, 45, 90 and 135 minutes p.m.) are given. Each mean value and the corresponding standard deviation were calculated from the results obtained on 7 rats. The upper standard deviation overlaps the lower standard deviation of the mean value. This corresponds to an overlap of about 45% of the time interval between two measuring points (45 minutes time interval; overlap of approximately 20 minutes). The 95%-limits of confidence of the mean chronaxies would therefore cover nearly the whole period investigated. Unfortunately the single calculated values of the chronaxies of 7 rats at 4 different postmortem intervals are not communicated.

With this standard deviation in the first 90 min p.m., the p.m. rise of chronaxie will only be of very limited value in determining the time since death in the early pmi, especially since the experimental findings were obtained under controlled and standardized circumstances. The range of scatter of chronaxies will of course increase under different circumstances (environmental temperature, mode of death etc.).

The hope of the authors that the chronaxie may be useful for a longer period in humans than in rats is not really justified.

The resuscitation period of mammalian peripheral nerve is about 30–35 minutes (Gerard 1930; Wright 1946). The supravital period of peripheral nerves outlasts the resuscitation period for some time (Madea and Henßge 1991). According to several different authors the p.m. duration of excitability of peripheral nerves is relatively homogenous in rats and humans (Nokes et al 1991; Sliwka and Bloch 1988; Krause et al. 1976) with a tendency to be longer in humans than in rats.

The analogous conclusion of the authors that the duration of excitability of peripheral nerves would last proportionally longer in human bodies as the body cooling (rectal temperature) is prolonged compared to rats is completely wrong. If any, a very low influence could be theoretically expected but in the opposite direction. The cooling velocity of central axial measuring points depends mainly on the diameter of the body region where temperature probes are taken. The lower the diameter, the faster central axial temperatures will cool down to ambient temperature (Sellier 1958). Since the human thigh has a greater diameter and will consequently take a longer time to cool down, the metabolic processes will run down at a higher rate until energy reserves are exhausted according to van't Hoff's rule. Therefore the excitability of the human sciatic nerve could be somewhat shorter than in rats. The statement of the authors that "methods which are in use for estimating the postmortem interval are unreliable, inaccurate and only useful in approximately the first 18 h after death" are not in accordance with the recent literature on time of death estimation (Henßge 1988; Albrecht et al. 1990; Madea and Henßge 1990a; Henßge et al. 1988; Marshall and Hoare 1962; Marshall 1962a, b). The claim of the authors "to find some time-related quantity which changes after death in an accurately measurable way" is not new (see experiments on postmortem rise of galvanic threshold by Joachim 1976, Joachim and Feldmann 1980; Madea and Henßge 1990b and other criteria of muscular contraction after electrical stimulation; Henßge et al. 1984; Madea 1989; Madea 1992a, b).

The accurate measurement of "some time related" quantity "which changes after death" does not solve the problem of the interindividual variability; this becomes

evident from the authors' own results for the first 90 minutes pm! This interindividual variability is expressed in statistical terms (standard deviation, 95%-limits of confidence). In so far there is no difference between the authors' results and those methods "which are in use for estimating the postmortem interval" and which are "unreliable and inaccurate".

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Prof. Dr. med. B. Madea
Institut für Rechtsmedizin der Universität zu Köln
Melatengürtel 60–62
W-5000 Köln
Germany

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Reply

Dear Sir,

Thank you for the copy of Professor Madea's letter concerning our article entitled "Nerve conduction as a means of estimating early post-mortem interval" in volume 105 (pp 69–74) of this Journal.

In the first instance we have to acknowledge that a typographical error was unfortunately not corrected in the galley proofs of this article. This refers to the length of duration of stimuli as 0.5 to 4 us which units should have read as being *ms*. Nevertheless although this correction had not been made an examination of Figures 5 and 6 of our paper shows that the curves become asymptotic at values between about 0.3 and 0.8 ms. On a "log-millisecond" scale which would correspond to a value of about 2–6 ms.

Prof. Madea is not perhaps entirely correct to state that the value of rheobase is calculated and not measured. Previous work by one of the co-authors of our paper which was referred to in the paper itself (Glasby et al. 1988) supports this.

For each rat an individual S. D. curve was constructed while the animal was alive and at the various times when the measurements were taken post-mortem. All of these curves became asymptotic within the above range of stimulus durations. In order to compare curves, the individual values of stimulus strength are normalised by dividing each by the established rheobase for that rat. This allows a more accurate comparison. The mathematical basis for this has been discussed in the paper by Glasby et al. (1988) already referred to. In the book by Bayliss (1960), Lapique in his original appreciation of the S. D. curve plotted "strength in terms of the rheobase as unit". It would be impossible to claim a firmer precedent.

Prof. Madea also appears to be suggesting that the S. D. curve for skeletal muscle is the same as that for nerve: it has to be pointed out that we were using nerve and not muscle for our experiments. The S. D. values which he quotes for muscle are in no doubt and indeed for skeletal muscle the stimulus duration needed to determine rheobase is about 100–500 ms. Equally for nerve the stimulus duration needed is about 3–5 ms. (Davson and Eggleton 1968). We have found in many instances that the curve for the rat's sciatic nerve is asymptotic within the stimulus range that we have used. Prof. Madea's very long stimulus duration may be explicable by his choice of electrodes. He does not state how he stimulates and records from the muscle, but it is well known that the rate at which current spreads will affect this measurement. We have stimulated and recorded with bipolar electrodes placed quite close together which is acknowledged to define more closely the region of nerve under test and to be the most consistent and best way for measuring the nerve strength duration relationships. With all animal experiments extrapolation of results from the animal to the human situation may be very difficult and fraught with problems.

The hypothetical statement that we have made in the "discussion" section of our paper in relation to the relative rates of post-mortem cooling between the rat and man, remains to be proved. Experiments will have to be conducted by attaching probes directly to human nerves as was done with our experimental animals. This would avoid any interference by intervening tissues into the measurements conducted.

It has to be accepted that in our paper, the standard errors of chronaxie are such, given the limited numbers of animals in our experiments, that there is an overlap of the means. This point was specifically referred to in the paper because of specific editorial comment prior to the acceptance of the paper for publication. It was not felt to be necessary to include all the individual measurements as otherwise the figures would have too cumbersome and the text too lengthy.

Publications on studies on post-mortem nerve and muscle excitability have appeared in the international literature for over three decades. Our experimental studies were intended to be a further small contribution to the wealth of studies already in print.

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M. A. Glasby
Department of Anatomy
A. Busuttil
Forensic Medicine Unit, Department of Pathology
University of Edinburgh, Scotland