Obstetric brachial plexus lesions

To The Editor: We would like to comment on the paper by Pondaag et al. (Pondaag W, van der Veken LPAJ, van Someren PJ, et al: Intraoperative nerve action and compound motor action potential recordings in patients with obstetric brachial plexus lesions. J Neurosurg 109:946–954, November, 2008). We have only operated on 16 neonates with obstetric brachial plexus lesions (OBPL) taken from a group of 171 who were referred for possible surgical reconstruction. The majority of these patients showed substantial clinical improvement without surgery over the course of many months after birth and were not candidates for surgery. In addition, we have operated on a large number of adult patients with stretch/avulsive injuries. We agree that there are significant differences between infant brachial plexus stretch injuries due to birth OBPL and adult stretch/contusive lesions to the plexus. However, we do not believe that the underlying pathophysiological mechanisms are so different that operative recordings would be invalidated in one scenario and not the other. Based on our own limited experience of neonatal OBPLs combined with a more extensive experience in the adult population, we have determined that there are 5 major categories of recording patterns in stretch injuries and operative neurophysiological findings.

Recording Patterns in Stretch Injuries

1) Normal nerve action potential (NAP) is recorded from a normal spinal element and its outflow (often C-7 or C-8). Compound motor action potentials (CMAPs) with good amplitude of response and conduction velocities of 50–70 m/second are observed.

2) Small and slowly conducted NAPs are seen in an injured spinal element with continuity and good regeneration (axonotmesis, Sunderland Grade II or III). A regenerative NAP is usually small and of low amplitude, has slow conduction (20–40 m/second) and is usually distinct from the stimulus artifact. The response is always reproducible on sequential recordings. There may or may not be an evoked CMAP depending on the time between injury and when the recording is done. Sometimes, it will be positive in OBPLs for biceps or triceps after 9 months or more if the lesion to the element was complete to begin with, or of course sooner if the injury was incomplete to begin with.

3) Normal-appearing NAPs are recorded with avulsion that is purely preganglionic. There is an NAP with good amplitude and conduction velocity of a normal or even greater velocity. Evoked CMAPs are usually absent unless stimuli spread to adjacent elements without preganglionic injury. This NAP is positive because the injury is solely to sensory axons prior to the dorsal root ganglion (DRG). As a result, the wallerian process does not affect those axons beyond the DRG or along any of their more distal course. This NAP can be distinguished from a regenerative one by its larger amplitude and much faster conduction either at or above normal limits and especially by the fact that one can record it well distal to the clavicle (for example, from musculocutaneous, axillary, or radial nerves) at a point in time (3–12 months postinjury) when sufficient new axons and thus a regenerative NAP could not have been expected to reach those levels. The CT myelogram or MR image is often positive for a meningocele or other abnormality but not always. Of great importance, exploration, even at a foraminal level, does not always show grossly intact axons and/or DRGs. Inspection of spinal nerves and trunks may appear intact, but not always because there may be a degree of more lateral injury, although not enough to completely destroy the spinal sensory axons.

4) A flat NAP trace can be due to pre- and postganglionic injury in which, despite the preganglionic injury, there is a neuroma-in-continuity involving the spinal nerve, trunk, or even divisions. Because of the postganglionic portion of the injury, there is no or little preservation of axons, even the sensory ones, and thus no conducted NAP. There is no evoked CMAP unless adjacent or nearby less involved elements have stimuli transmitted to them and thence to muscle. Sectioning proximally on such a spinal nerve usually shows either gross evidence of avulsion or heavy scar, and in any case it is not a useful structure from which to lead out grafts.

5) A flat NAP trace can be due to a neurotmetic (Sunderland Grade IV) lesion-in-continuity lateral to the root at spinal nerve and upper trunk levels. Evoked CMAPs will usually be absent unless less involved elements with connection(s) to muscle(s) being tested are stimulated. In the occasional case, a small number of fibers may successfully penetrate the lesion and reach muscle. However, the flat NAP trace produced by insufficient numbers of regenerating axons is not likely to be followed by a successful return of adequate function. In fact, these few regenerated fibers may provide a misleading impression when they are all synchronized by stimulus pulses and produce visible muscle contractions or a clear CMAP. It takes only a few fibers reaching muscle to evoke a recordable CMAP or even to mediate a visible contraction.

There needs to be recognition of some of these differences when data are presented about plexus stretch lesions. This is especially so for Items 3 and 4 above. Failure to recognize these differences could lead to mistaken analysis. For example, a flat trace across a lesion could, if the lesion is lateral to the root, be due to neurotmesis and lead to resection and repair with some hope of recovery as recognized by the authors.

On the other hand, if the flat trace is due to a pre- as well as postganglionic lesion, resection and direct repair will be of no value. A CT myelogram will not always be abnormal at such levels. Neurolysis of an element conducting an NAP due to retained sensory fibers because of a preganglionic lesion with some more lateral damage also does not result in function. Timing is also impor-
tant. Neurolysis without resection of an element having a conducted NAP and distal CMAPs at a year or more postinjury may not suffice.

In the 16 cases of OBPL at Louisiana State University Health Sciences Center that were surgically treated because of poor biceps and/or shoulder abduction ≥ 10 months after birth, NAP studies were performed on exposed elements and 96 recordings were made. This resulted in 30 elements having a neurolysis due to positive recordings, 19 having grafts due to negative traces in which there was no operative evidence of avulsion, and normal preoperative CT myelograms at those levels. Each of the resected specimens was Sunderland Grade IV histologically. Ten nerve transfers were due to preganglionic or pre- and postganglionic traces combined with surgical inspection. Usually, but not always, there were abnormal CT myelographic findings at those levels.

**Surgical Inspection**

It is claimed that one can tell whether a root is avulsed in the operating room, and such is the case when DRG is seen outside the foramen. However, there can be significant preganglionic avulsion without such a finding even if the CT myelogram is negative at that level. Then, if the surgeon is unaware of what a preganglionic NAP looks like, he or she will assume that there are good regenerative axons and axonotmesis or sparing and perform neurolysis on an element in which, because of more proximal irreversible injury, there is no hope of recovery. Purely, preganglionic lesions are not always accompanied by normal-appearing distal elements, although such is often the case. Distal elements can be thickened and swollen and still contain preserved sensory fibers. The more severe axonotmetic lesion (especially a Sunderland Grade III) can be swollen and firm and appear quite scarred on gross inspection, causing difficulty in discerning the fascicular continuity, just as the neurotmetic one (Sunderland Grade IV). The ability to determine fascicular continuity by gross inspection, even with magnification but without internal neurolysis is, in the typical neurome, impossible. Even with internal neurolysis, fascicular continuity through the lesion does not insure recovery because fascicular damage itself may be neurotmetic, especially with stretch injuries.

**Technical Considerations**

As pointed out by the authors, the infant plexus is small and relatively short, so direct recordings are technically difficult and need to be very precise. However, such recordings are less useful when the time base is > 0.5 msec/division. In the recordings in this paper, the time base for NAPs was 2 msec/division. Because the study was done in infants, only a 3–4-cm distance was used for stimulation and recording studies. That setting does compress the stimulus artifact, but it also makes it more likely that an NAP will be enfolded within the stimulus artifact.

When relatively short distances are used for recording, as was the case here, the time base should be set on 0.5 msec/division or at most, 1 msec/division, but not a 2 msec/division except when stimulating and recording over longer distances. Stimuli also need to be brief (≤ 0.05 msec in duration) not only to reduce stimulus artifact, but also because longer-duration stimuli can activate small, poorly myelinated fibers that may or may not eventually become large enough to produce useful function.

The authors claim that the waveform marked initially in Fig. 2A is an NAP. If so, that would have a latency of 4 msec and if recorded at 3 cm a velocity of only 7.5 m/second and, if recorded at 4 cm, only 10 m/second, so it is not a good example of an NAP, especially in the later months after the birth injury when these recordings were done. But is it really an NAP? It is more likely to be stimulus artifact overrun and not an NAP. What follows the NAP shown would, if it is truly an NAP, be 6 m/second if recorded at 4 cm and 4.3 m/second if recorded at 3 cm. If the 60-cycle notch filter was left on on the recording machine, such a false response and the waves that follow could be recorded. That secondary wave could also be a muscle action potential (MAP) picked up by the NAP recording electrodes in some settings; such an MAP is possible when muscle is directly recorded and yet there is an absent NAP. This is because it only takes a few fibers reaching muscle to evoke a recordable CMAP, but it takes several thousand moderate-sized fibers across a lesion to record a true NAP. Thus, in normal nerves, 18 of 18 recordings were positive as seen in their Table 3. In lesions believed to be “axonotmetic” on inspection, despite the difficulties of being certain of that diagnosis, 112 of 114 traces were positive, so in that setting, NAPs appeared to be at least, as the authors found, accurate.

The NAP recordings appear to fail in their hands in what they describe as “neurotmetic” lesions (53 of 373 lesions had positive NAPs), whereas the others were flat, fitting better with a neurotmetic lesion. Were some of these “neurotmetic” lesions falsely resected Sunderland Grade III lesions in which the element can be swollen and scarred? Their text suggests yes. Perhaps some of these lesions had a preganglionic as well as a postganglionic axonotmetic lesion and were swollen and scarred lateral to the foramen. When a recording is classified as absent, it may mean either that the CMAP was absent biologically or that it could not be recorded. To exclude the latter, it is often helpful to record from a segment of known normal nerve or plexus element. This ensures that all technical considerations are present for a successful recording. Unfortunately, the authors emphasize their observation that 28 of 62 traces on elements believed to be more proximally avulsed had positive NAPs. However, this is exactly what one would expect because a significant proportion of avulsions, not only in adults, but also in infants, are purely preganglionic and thus have distal sensory fiber sparing and therefore paradoxically have a positive NAP, which is possible despite a negative myelogram at that level.

**Differences Between Birth and Adult Stretch Palsies**

Despite these observations, we agree that there is no question that most infant brachial plexus stretch injuries due to birth OBPL differ from adult stretch/contusive lesions to the plexus. The infant palsies are usually less se-