Preliminary Results of the Australasian Regional Anaesthesia Collaboration

A Prospective Audit of More Than 7000 Peripheral Nerve and Plexus Blocks for Neurologic and Other Complications

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Background and Objectives: Peripheral nerve blockade is associated with excellent patient outcomes after surgery; however, neurologic and other complications can be devastating for the patient. This article reports the development and preliminary results of a multicenter audit describing the quality and safety of peripheral nerve blockade.

Methods: From January 2006 to May 2008, patients who received peripheral nerve blockade had data relating to efficacy and complications entered into databases. All patients who received nerve blocks performed by all anesthetists during each hospital's contributing period were included. Patients were followed up by phone to detect potential neurologic complications. The timing of follow-up was either at 7 to 10 days or 6 weeks postoperatively, depending on practice location and time period. Late neurologic deficits were defined as a new onset of sensory and/or motor deficit consistent with a nerve/plexus distribution without other identifiable cause, and one of the following: electrophysiologic evidence of nerve damage, new neurologic signs, new onset of neuropathic pain in a nerve distribution area, paresthesia in relevant nerve/plexus distribution area.

Results: A total of 6950 patients received 8189 peripheral nerve or plexus blocks. Of the 6950 patients, 6069 patients were successfully followed up. In these 6069 patients, there were a total of 7156 blocks forming the denominator for late neurologic complications. Thirty patients (0.5%) had clinical features requiring referral for neurologic assessment. Three of the 30 patients had a block-related nerve injury, giving an incidence of 0.4 per 1000 blocks (95% confidence interval, 0.08–1.1:1000). The incidence of systemic local anesthetic toxicity was 0.98 per 1000 blocks (95% confidence interval, 0.42–1.9:1000).

Conclusions: These results indicate that the incidence of serious complications after peripheral nerve blockade is uncommon and that the origin of neurologic symptoms/signs in the postoperative period is most likely to be unrelated to nerve blockade.

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Peripheral nerve and plexus blockade (PNB) is associated with excellent patient outcomes after surgery. These benefits include superior postoperative analgesia and recovery compared with general anesthesia^{1,2} and opioid analgesia,³ and similar quality of analgesia compared with epidural analgesia.⁴ Although considered rare, neurologic and other serious complications after PNB can be devastating to the patient. The results of the largest study performed to date support the rarity of serious complications after PNB,⁵ although the reported incidences vary significantly.⁶ This is in part due to variability in the methods used to capture anesthesia-related neurologic complications. These methods have included direct follow-up by the anesthesiologist, patient self-reports, surgical follow-up, and voluntary surveys. Once a potential complication is detected, there is also variability in both the pathway of neurologic investigation and definition for injury.⁶ In addition, determining the incidence of rare adverse events requires data from a large number of patients. The Australasian Regional Anaesthesia Collaboration (ARAC) has been established so that data describing the quality and safety of PNB from tens of thousands of patients can be collected and analyzed. The objective of this article is to describe the development and report the preliminary results of this audit. In addition, the methods used to capture and investigate potential complication are detailed.

METHODS

The human research and ethics committee of each hospital contributing to this project has either approved this project as a quality-assurance activity or low-risk research, or formally waived the requirement for approval. During the calendar year 2006, data relating to PNB performed at 2 hospitals were prospectively entered into proprietary databases (Microsoft Access 2003; Microsoft Corporation, Redmond, Wash; and SPSS Statistics; SPSS Inc, Chicago, Ill). During this period, an online open source database (MySQL; MySQL Inc, Cupertino, Calif) was developed by one of the authors (R.D.T.), recognizing that the Web-based interface would simplify data entry and facilitate multicenter collaboration. From January 2007 to May 2008, a further 7 hospitals/practices contributed data to this project, with most data entered directly into the online database.⁷

All patients who received PNB for anesthesia and/or analgesia performed by all anesthetists during each hospital's or practice's contributing period had their PNB recorded and were systematically followed up for potential neurologic complications using direct contact by phone. Appendix 1 lists the hospitals/practices that contributed to this project. Data were recorded relating to the performance and efficacy of PNB,

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Australasian Prospective Audit of PNBs

adverse effects, and complications. These data included a unique patient code, date of procedure, surgery type, needle bevel type, local anesthetic and dose, level of sedation, and block success. Peripheral nerve and plexus blockade type was recorded: interscalene, periclavicular, axillary, distal humeral/forearm, femoral/fascia iliaca, sciatic, and other peripheral lower limb nerves and trunk blocks. The technology used to locate plexus/ nerves was recorded: ultrasound alone, nerve stimulator alone, ultrasound and nerve stimulator, and other.

Appendix 2 lists the definitions for block success and immediate complications. The definitions used for these categories were obtained from published guidelines.⁸ The definitions used for this project were also available online at www.regional.anaesthesia.org.au. The timing of follow-up for potential neurologic complications was either at 7 to 10 days or 6 weeks postoperatively, depending on practice location and time period. Patients were not considered to be uncontactable by phone until 4 attempts had been made at different times and using alternative phone numbers if available. The denominator used to determine the incidence of neurologic injury was calculated from the number of procedures performed on the number of patients successfully contacted.

To detect potential neurologic complications, patients were asked a standardized set of questions: Do you have any numbness? Do you have any tingling? Do you have any abnormal sensations? Do you have any pain? Do you have any weakness? These questions were asked in relation to the operative limb, and if the patient responded yes to any of the questions, then further queries were made taking into account the anatomy relevant to the surgery and the peripheral nerve/plexus block. Symptoms that were immediately adjacent to the wound, consistent with normal tissue healing or the initial trauma, were not considered relevant in terms of anesthesia being a causal factor. For patients with ambiguous symptoms or complaints, repeat contact was made with the patient. Triggers for referral to a neurologist were new onset of motor and/or sensory deficit, nonresolving paresthesia, pain, allodynia, or dysesthesia and any concern expressed by the surgical team regarding the potential for an anesthetic-related neurologic deficit. Assessment by the neurologist consisted of history, examination, documentation, and investigation when appropriate. Investigations included electrophysiology (nerve conduction studies [NCSs] and/or an electromyography [EMG]),

imaging (computed tomography [CT], magnetic resonance imaging [MRI]), and blood tests. Late neurologic deficits related to anesthesia were defined as a new onset of sensory and/or motor deficit consistent with a nerve/plexus distribution without other identifiable cause and one of the following: electrophysiologic evidence of nerve damage, new neurologic signs, new onset of neuropathic pain in a nerve distribution area, or paresthesia in relevant nerve/plexus distribution area. Long-term neurologic deficit was defined as the criteria for late neurologic deficit having been met and with persistence of symptoms for longer than 6 months after onset.⁸

All statistical analyses were performed using Stata 8.2 (StataCorp, College Station, Tex). In the results, data that are normally distributed are presented as mean (SD), and nonnormally distributed and/or skewed data are presented as median (10th-90th percentiles). Adverse events are expressed as n/1000 and 95% confidence intervals (CIs). The 95% CIs for adverse events were calculated using a Poisson distribution.

RESULTS

During the study period, a total of 6950 patients received 8189 peripheral nerve or plexus blocks. Table 1 lists the block type, success rate, and timing of follow-up at each hospital. Of the 6950 patients, 6069 were successfully followed up. In these 6069 patients, there were a total of 7156 episodes of PNB forming the denominator for late neurologic complications. Table 2 summarizes PNB characteristics including level of sedation during block performance, technologies used to locate nerves (and relevant success rates), local anesthetic dosages, needle type, and catheter usage. Ultrasound imaging was used to locate nerves/plexuses in 63% of procedures.

Thirty patients (0.5%) had clinical features requiring referral for neurologic assessment. Clinical details of patients referred for neurologic assessment are summarized in Table 3. Three of the 30 patients referred met the criteria for nerve injury due to PNB. giving an incidence of 0.4 per 1000 PNBs (95% CI, 0.08-1.1:1000). In 2 of the 3 patients with nerve injury related to PNB, nerve stimulation alone was used to locate nerves, and in the remaining patient, combined ultrasound and nerve stimulation was the technique used. The patients with nerve injury due to PNB are listed in Table 3 as patients 6, 27, and 30, with sensory

TABLE 1. DIOCK Type, Success Rate, and Titting OF FONOW-OP at Each Hospit	TABLE 1.	Block Type,	Success Rate,	and Timing	of Follow-U	p at Each Hospi
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Hospital	ISB	РСВ	AXB	UL	Trunk	FNB	LP	SCI	LL	Other	Total	Success, %	7–10 d/6 wk/Total Follow-Up, %
A	13	18	16	3	30	38	0	14	2	0	134	93	49/28/78
В	79	7	52	5	31	58	0	27	2	0	261	95	21/58/79
С	21	10	1	4	44	84	0	13	0	0	177	94	16/54/70
D	31	15	12	3	270	76	0	27	2	1	437	89	41/45/86
E	15	7	3	0	3	60	29	68	1	1	187	99	66/15/81
F	31	16	17	3	37	95	12	54	6	3	274	97	12/57/69
G	476	242	81	0	67	1061	645	565	0	61	3198	86	90/0/90
Н	207	167	1039	189	183	890	5	505	64	30	3279	89	31/61/92
Ι	16	17	30	4	46	101	0	12	6	10	242	96	0/46/46
Total	889	499	1251	211	711	2463	691	1285	83	106	8189	89	53/34/87

Data are presented as n (number of blocks) or percentage (%). Success was defined as block with successful puncture and injection of local anesthetic with development of anticipated block characteristics including evidence of sensory or motor block. Six-week follow-up or 7- to 10-day follow-up refers to patients successfully followed up at these time periods. Total percentage may not equal 100 due to rounding.

ISB indicates interscalene block; PCB, periclavicular block; AXB, axillary brachial plexus block; UL, distal upper limb block; FNB, femoral nerve or fascia iliaca block; LP, posterior lumbar plexus block; SCI, sciatic nerve block; LL, distal lower limb block.

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Sedation during block ($n = 8189$)	
Alert	52%
Sedated	15%
Unresponsive*	23%
Not recorded	10%
Technology used to locate nerve/plexus rates (%) \dagger (n = 8189)	and reported success
Ultrasound	13% (93)
Nerve stimulator	30% (92)
Ultrasound and nerve stimulator	50% (86)
Other	7% (91)
Local anesthetic $(n = 4991)$	
Ropivacaine	55%
Lidocaine	9%
Bupivacaine	3%
Other	32%
Local anesthetic dosage, mg/kg	
Ropivacaine	2.03 (0.93) (n = 2236)
Lidocaine	4.80 (2.38) (n = 353)
Bupivacaine	1.23 (1.09) (n = 81)
Needle type $(n = 4965)$	
Short bevel	92%
Hypodermic	3%
Other	4%
Not recorded	1%
Catheter (n = 5286)	
Yes	22%
No	77%
Not recorded	1%

TABLE 2. Peripheral Nerve Block Characteristics

* Unresponsive includes block performance after general or neuraxial anesthesia.

†Success was defined as block with successful puncture and injection of local anesthetic with development of anticipated block characteristics including evidence of sensory or motor block, calculated from a cohort of 5984 procedures. Total percentage may not equal 100 due to rounding. Data are presented as percentage (%) or mean (SD).

deficits of duration less than 6 months, 12 months, and greater than 6 months, respectively. The remainder of the patients referred for neurologic assessment (27/30) had postoperative symptoms/signs that were unrelated to PNB.

Table 4 lists the clinical details where PNB was complicated by local anesthetic toxicity. The overall incidence of this complication was 0.98 per 1000 PNBs (95% CI, 0.42– 1.9:1000). The estimates of both immediate and delayed complications according to nerve localization techniques are listed in Table 5. There were no complications recorded from the following categories: unintentional puncture of an adjacent organ, respiratory depression/arrest, pneumothorax, cardiac arrest, or death.

DISCUSSION

This prospective audit includes the follow-up of 7156 PNBs in 6069 patients, resulting in an incidence of late neurologic deficit of 0.4 per 1000 blocks. The strength of this project is its robust neurologic follow-up including direct follow-up of patients and actively seeking patients with potential complications. This approach is associated with more reliable capture of complications compared with a passive approach.9 Additional strengths of this study are the standardized questionnaire for detecting potential complications, the neurologic referral pathway, and definition for nerve injury due to PNB. Figure 1 summarizes the neurologic follow-up pathway, and Table 3 shows the results of investigations performed for this report. It was only by evaluating patients with a focused history and examination and performing NCSs, EMG, and imaging studies (CT, MRI) that we were able to separate PNB causes of injury from those unrelated to PNB. Patients who met the criteria for referral to a neurologist were 9 times more likely to have a cause unrelated to PNB than they were to have symptoms/signs attributable to PNB. Without careful evaluation, a patient's postoperative neurologic features can be incorrectly attributed to regional anesthesia. The potential for these scenarios is similar to obstetric anesthesia where regional anesthesia is often blamed but rarely responsible.¹⁰

In the largest surveys with a denominator of more than 70,000 patients, Auroy et al^{5,11} reported an incidence of nerve injury related to anesthesia of 0.02%, similar to our incidence of 0.04%. Capdevila et al¹² reported an incidence of nerve injury of 0.21% after continuous peripheral nerve blockade, with all deficits resolved by 10 weeks. Although the rate of complications reported by Capdevila et al¹² is increased compared with that of Auroy et al^{5,11} and our current results, this is explained by the methodology used by Capdevila et al¹² including follow-up of patients 24 hrs postoperatively and a definitive pathway of investigation including EMG performed within 6 to 12 hrs of observing a neurologic deficit. In our study, the pathway of investigation was defined, but its activation was delayed, and therefore, our incidence of complications is lower. The results reported by Auroy et $al^{5,11}$ and Capdevila et al^{12} are from PNB using nerve stimulation to locate nerves, whereas this current report has occurred during the emergence of ultrasound-guided PNB, where 63% of blocks were performed using ultrasound technology. Despite a renewed interest in PNB and changes in technique over the last decade, the reported incidence of nerve injury related to PNB has not changed. This may be due to the difficulty in detecting reductions in complications that only occur infrequently. Because of the low numbers of complications, our results are not able to establish if ultrasound technology confers safety benefits compared with nerve stimulation. Theoretically, ultrasound imaging should prevent direct needle trauma to a nerve. However, there are limitations with ultrasound technology including its low native resolution, limited plane of view, and its operator-dependent image quality.

In this report, the incidence of systemic local anesthetic toxicity was 0.98 per 1000 blocks. This incidence is similar to the 0.08% reported in the survey of Auroy et al¹¹ and parallels the lack of change in the reported incidence of nerve injury over time. Local anesthetic toxicity occurred despite the utilization of ultrasound guidance in 50% of patients with reported toxicity. Theoretically, real-time imaging of the needle and vascular structures provides a mechanism to avoid intravascular injection and systemic toxicity, and in both this report (Table 5) and a recently published meta-analysis, there is a reduced risk of vascular puncture using ultrasound guidance compared with nerve stimulation.¹³ Despite this, local anesthetic toxicity has been reported with ultrasound guidance.¹⁴ The limitations of both ultrasound technology and its operators in reducing nerve injury are also relevant in reducing the incidence of systemic local anesthetic toxicity. For example, local anesthetic injection may occur out-of-plane, potentially preventing early warning of intravascular injection. Furthermore, toxicity may occur secondary to delayed tissue absorption, highlighting the importance of noting the recommended maximal dosage of a local anesthetic,

Patient	Hospital	Surgery	PNB	Presentation	NCS	MRI	Results of Investigations	Comments
1	Н	Biopsy humerus	ISB	Motor deficit, wrist drop	\checkmark	×	NCS/EMG abnormal humeral injury	Surgical cause
2	Н	Rotator cuff repair	ISB	Pain	\checkmark	\checkmark	NCS/EMG normal	CSD, full recovery
3	Н	Acromioplasty	ISB	Paresthesia	\checkmark	\checkmark	NCS/EMG/MRI normal	Unrelated to PNB
4	Н	Shoulder arthroplasty	ISB	Paresthesia	\checkmark	×	NCS normal	Unrelated to PNB
5	G	Acromioplasty	ISB	Paresthesia	\checkmark	×	NCS normal	Unrelated to PNB
6	G	Rotator cuff	ISB	Paresthesia/ dysesthesia	×	×	Inflammatory plexopathy	Related to PNB
7	G	Subacromial decompression	ISB	Paresthesia/pain	\checkmark	×	NCS—distal deficit, neurapraxia	Unrelated to PNB
8	Н	AV fistula	PCB	Paresthesia	\checkmark	\checkmark	NCS normal, MRI normal, MRI—CSD	Unrelated to PNB
9	Н	ORIF humerus	PCB	Paresthesia	\checkmark	×	NCS/EMG—ulnar nerve, neuropathy fracture site	Symptoms due to fracture
10	G	Carpal tunnel	PCB	Paresthesia	\checkmark	×	Ulnar entrapment	Unrelated to PNB
11	А	AV fistula (3)	PCB	Paresthesia/pain	\checkmark	×	Antebrachial neuropathy	Unrelated to PNB
12	Н	Wrist fusion	AXB	Pain	\checkmark	×	NCS normal	Unrelated to PNB
13	Н	Scaphoid excision	AXB	Paresthesia	\checkmark	\checkmark	NCS, CTS, MRI normal	Unrelated to PNB
14	Н	Removal plate	AXB	Paresthesia	\checkmark	\checkmark	NCS/EMG normal MRI—CSD brachial plexus normal	Unrelated to PNB
15	Н	Amputation finger	AXB	Paresthesia	\checkmark	\checkmark	Ulnar neuritis	Unrelated to PNB
16	Н	Hand arthroplasty	AXB	Pain	\checkmark	\checkmark	CTS, ulnar neuropathy, CSD (MRI)	Unrelated to PNB
17	Н	Wrist synovectomy	AXB	Paresthesia	\checkmark	×	NCS normal	Unrelated to PNB
18	Н	ORIF scaphoid	AXB	Pain	\checkmark	×	NCS normal	Unrelated to PNB
19	Н	Thumb suspensioplasty	AXB	Paresthesia	\checkmark	×	Ulnar neuropathy	Unrelated to PNB
20	G	TKA	LP	Paresthesia	×	×	Refused EMG, CPN injury	Unrelated to PNB
21	G	TKA	LP	Motor	\checkmark	×	NCS—CPN injury	Unrelated to PNB
22	Н	TKA	FNB	Paresthesia, weakness	\checkmark	\checkmark	Lumbar canal stenosis, DN	Unrelated to PNB
23	Н	TKA	FNB	Paresthesia	\checkmark	×	NCS normal	Unrelated to PNB
24	Н	TKA	FNB	None	\checkmark	\checkmark	NCS/EMG mildly abnormal	FNB or tourniquet neurapraxia
25	Н	TKA	FNB	Paresthesia/pain	\checkmark	×	NCS-DN, radiculopathy	Unrelated to PNB
26	Н	AKS (2)	FNB/SCI (2)	Paresthesia	\checkmark	×	NCS—bilateral neuropathy normal	Underlying neuropathy
27	G	AKS	FNB/SCI	Paresthesia	×	×	Refused NCS	Related to PNB
28	G	AKS	FNB/SCI	Paresthesia/pain	×	×		Unrelated to PNB
29	G	ORIF calcaneus	SCI	Motor (foot drop)	\checkmark	\checkmark	NCS/EMG—CPN injury	Unrelated to PNB
30	D	Foot surgery	SCI	Paresthesia	\checkmark	×	Abnormal NCS	Related to PNB

TABLE 3. Patients Referred to Neurologists and Results of Investigations

PNB indicates peripheral nerve or plexus blockade; ORIF, open reduction internal fixation; $\sqrt{}$, investigation performed; \times , no investigation; ISB, interscalene block; PCB, periclavicular block; AXB, axillary brachial plexus block; FNB, femoral nerve block; AV, arteriovenous; LP, posterior lumbar plexus block; SCI, sciatic nerve block; CTS, carpal tunnel syndrome; CSD, cervical spine degeneration; TKA, total knee arthroplasty; AKS, arthroscopic knee surgery; CPN, common peroneal nerve; DN, diabetic neuropathy.

taking into account the age, weight, and comorbidities of the patient; the site of injection; and pregnancy.¹⁵

This project has several potential and real limitations. Postoperative follow-up was performed at either 7 to 10 days or 6 weeks. Follow-up at 6 weeks, as occurred in 34% of our patients, may have missed a temporary nerve injury presenting in the early postoperative period. The influence of timing of follow-up is illustrated in several reports^{16–21} where the rate of early deficit was high but temporary, with almost all patients having

complete recovery. Symptoms reported by patients in the early postoperative period may also represent tissue healing. Alternatively, if patients have swelling or postoperative pain, they may not notice features of nerve injury and may present later.⁹ There is no ideal time period to follow up patients to detect potential neurologic complications. However, from 2008 onward, this project follows up patients at 7 days postoperatively because NCSs performed relatively early in the postoperative period may document a preoperative neuropathy.

	Surgery Site/ Hospital	Nerve Location Technique	PNB	Local Anesthetic	Dose, mg/kg	Comments	Signs/Symptoms
Major t	oxicity						
1	Knee/I	NS	FNB	Ropivacaine	2.1	IV injection, ST-segment depression	Tonic-clonic seizure
2	Hip/A	Landmark	FI	Ropivacaine	3.6	_	Tonic-clonic seizure
3	Hand/H	US + NS	AXB	Ropivacaine	1.8	IV injection, ropivacaine 69 µg/mL	Unconsciousness, tachycardia (adrenaline in injectate)
Minor	oxicity						
4	Finger/H	NS	AXB	Ropivacaine	1.2	IV injection	Disinhibition, auditory symptoms, agitation
5	Hand/H	US + NS	AXB	Ropivacaine	5.5	_	Agitation, successful block
6	Elbow/A	NS	ISB	Ropivacaine	2.7	IV injection	Tinnitus, drowsy, twitchy
7	Finger/H	US + NS	AXB	Lidocaine	7.5		Tinnitus, twitching
8	Hand/H	US + NS	AXB	Ropivacaine	4.5	Ropivacaine 3.1 µg/mL, amphetamine abuse	Mild CNS toxicity for 4–5 hrs

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PNB indicates peripheral nerve block; US, ultrasound; NS, nerve stimulator; FNB, femoral nerve block; IV, intravenous; FI, fascia iliaca block; AXB, axillary brachial plexus block; ISB, interscalene block,

A further limitation of this report is having 13% of patients without late follow-up. The authors believe that patients with complications would, more likely than not, present back to their original hospital with a complaint. In addition, the denominator for the incidence of nerve damage was calculated only from the number of PNBs performed in the number of patients successfully contacted. It is for these 2 reasons that we consider our denominator calculation accurate. Hospitals with low follow-up rates represent only one of the challenges of maintaining a large multicenter project.

A further challenge is ensuring that there is reliable data collection. The process we used to manage this was a combination of monthly emails highlighting key project requirements, use of local coordinators, and regular audits of the audit. These audits included monthly reviews of data entry at each contributing hospital and monthly reports. A potential limitation is that the criteria used to define late neurologic deficits included the proviso that there were no "other identifiable causes" for the patient's neurologic features in the postoperative period. This definition may result in exclusion of patients with preexisting conditions such as diabetes mellitus who may have a preexisting peripheral

neuropathy and theoretically be at risk for a new or progressive neuropathy.²² In this current study, these other identifiable causes were defined medical diagnoses made after postoperative assessment by a neurologist with expertise in peripheral neuropathies. Furthermore, the presence of preexisting conditions did not influence our decision to refer and investigate patients. For example, patient 22 (Table 3) had preexisting diabetic neuropathy and met the criteria for neurologic referral; however, the patient's clinical features and subsequent investigations were consistent with lumbar canal stenosis and not a nerve block-related injury. Where our results state that the clinical features were unrelated to PNB, an alternative diagnosis was made with well-defined clinical features and/or investigations; the results were either normal or not consistent anatomically with the PNB performed; or the criteria for late neurologic deficit (related to PNB) were not achieved for other reasons with these findings summarized in Table 3.

The imbalance of reporting is evident with different hospitals contributing varying numbers of both total and individual blocks. Operator expertise may be related to the volume of blocks performed²³; however, the frequency with

	Nerve L				
Complication	Nerve Stimulation (n = 2507)	Ultrasound (n = 5141)	Other (n = 541)	Total (n = 8189)	
Local anesthetic toxicity	1.2 (0.25–3.5)	0.8 (0.2-2.0)*	1.8 (0.05–10.3)	0.98 (0.42-1.9)	
Unintentional vascular puncture [†]	13.9 (8.2–21.9)	5.1 (3.0-8.1)‡	2.3 (0.06-12.8)	7.2 (5.1–10.0)	
Unintended paresthesia [†]	10.8 (5.9–18.1)	20.5 (15.9-25.9)*	2.3 (0.06–12.8)	16.8 (13.4–20.8)	
Late neurologic deficit	0.8 (0.1–2.9)	0.2 (0.005-1.1)*		0.4 (0.08–1.1)	
Long-term neurologic deficit	0.4 (0.01–2.2)	0.2 (0.05-1.1)*	_	0.2 (0.03-0.9)	

TABLE 5. Immediate and Delayed Complications According to Nerve Localization Technique

Data are presented as n/1000 (95% CI) procedures.

Ultrasound includes ultrasound used as the sole technology and combined ultrasound and nerve stimulation. Other comprises techniques not using nerve stimulation or ultrasound technology.

*Not statistically significant.

 \dagger Reduced total cohort (n = 4991), for nerve stimulation (n = 1297), ultrasound (n = 3260), and other (n = 434).

 \pm Indicates a statistically significant difference (P = 0.001; Poisson regression) between ultrasound and nerve stimulation and other techniques.



FIGURE 1. Pathway for neurologic follow-up, referral, and investigation.

which patients required neurologic referral was not disproportional in hospitals performing few blocks. The numbers of some block types are low, and in addition, lumbar plexus blockade was mostly performed at hospital G; therefore, we cannot calculate the incidence of injury for individual PNB types as previously published.⁵ Overall, the limited population of patients receiving PNB limits the generalizability of this report. More data providers and data collection will lead to future results being more generalizable. The overall proportion of patients who had their PNB performed after general or neuraxial anesthesia was 23% (with significant diversity between hospitals), and this may have resulted in underreporting of immediate complications such as minor local anesthetic toxicity and paresthesias. Patients who are unresponsive during PNB performance will be unable to report symptoms (pain and paresthesia) that may indicate impending nerve injury; hence, the American Society of Regional Anesthesia and Pain Medicine Practice Advisory on Neurologic Complications in Regional Anesthesia and Pain recommends that regional anesthesia blocks not be routinely performed in

adults with concurrent general anesthesia or heavy sedation.^{22,24} Because the audit involved collecting data from thousands of patients, it was not practical to systematically evaluate all patients for all complications. Therefore, for some immediate complications (eg, pneumothorax), their inclusion in the database would not have occurred unless clinically apparent. The standardized questionnaire used in this study to screen patients for nerve injury has an element of subjectivity; therefore, where patients had ambiguous symptoms or complaints, further contact was made with the patient.

An important aim of this current project was to determine the incidence of rare events and, in this regard, is similar to a proposal by the American Society of Regional Anesthesia and Pain Medicine to develop a large Postoperative Pain Database.²⁵ Obtaining reliable incidence data for neurologic injuries related to regional anesthesia is difficult, partly related to the infrequency with which these complications occur. Randomized controlled trials and other tools of evidenced-based medicine rarely exist and are unlikely to be available in the future.²² This collaboration has

been established, recognizing this issue. The Web-based interface facilitates ease of data entry, multicenter collaboration, and collection of data from a large patient cohort, as demonstrated by this report of 6950 patients. Collecting data from tens of thousands of patients is realistic, and we plan to continue this audit in 2010 and beyond. In the future, we aim to report on factors that may impact on the safety of PNB (including the technique used to perform PNB, level of sedation during block performance, operator experience, individual block types, patient comorbidities, barrier precautions, and infective complications), the quality of PNB (efficacy in relation to individual block types and operator experience, recovery parameters, and patient satisfaction), and trends in practice (eg, increased utilization of ultrasound technology, new techniques) and quantify the risk of serious complications with greater precision.

In conclusion, the results of the ARAC indicate that serious permanent complications after peripheral nerve/plexus blockade are uncommon and that the origin of neurologic symptoms/signs in the perioperative period is most likely unrelated to PNB. This report demonstrates proof of concept for both the online collection of data relevant to PNB and the subsequent direct patient follow-up to detect neurologic complications related to PNB. Larger patient samples are required to determine factors improving safety of PNB, and therefore, anesthesiologists regardless of practice type or size are invited to contribute to this collaboration.

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APPENDIX I

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APPENDIX II: THE DEFINITIONS FOR BLOCK SUCCESS AND IMMEDIATE COMPLICATIONS ARE AS FOLLOWS:

- 1. Successful block: successful puncture and injection of local anesthetic and with development of anticipated block characteristics including evidence of sensory or motor block.
- 2. Incomplete block failure: initially thought to be successful puncture and injection of local anesthetic and incomplete development of anticipated block characteristics but some evidence of sensory or motor block AND one of the following: insufficient surgical anesthesia requiring repeated block, insufficient requiring supplemental blocks, insufficient surgical anesthesia requiring supplemental systemic medication or conversion to general anesthesia, insufficient analgesia requiring repeated blocks, or insufficient analgesia requiring supplemental blocks.
- 3. Complete block failure: initially thought to be successful puncture and injection of local anesthetic and no development of anticipated block characteristics AND one of the following: no surgical anesthesia requiring supplemental blocks, no surgical anesthesia requiring supplemental blocks, no surgical anesthesia requiring supplemental systemic medication or conversion to general anesthesia, no analgesia requiring requiring supplemental blocks or no analgesia requiring systemic medication. If change in surgical plan/procedure is responsible for insufficient anesthesia/analgesia despite initially successful block, then do not code as block failure.

- 4. Successful general anesthesia required for other reasons: successful as for definition above, but general anesthesia planned for intraoperative period regardless of regional anesthesia.
- Successful spinal/epidural for other reasons: successful as for definition above, but spinal/epidural planned for intraoperative period regardless of regional anesthesia.
- 6. Minor systemic local anesthetic toxicity: either observed or suspected injection of local anesthetic into a vein or artery or suspected absorption of local anesthetic from injection site after injection AND three of the following features: agitation, anxiety, visual disturbances, acoustic disturbances, perioral paresthesia/numbness, dizziness, nausea, and muscle fibrillation/twitching.
- Major systemic local anesthetic toxicity: criteria for minor systemic local anesthetic toxicity are met AND one of the following seizure, somnolence, or loss of consciousness.
- 8. Paresthesia: definite symptom radiating in distribution of nerve.
- 9. Unintentional puncture of a major artery or vein: regional anesthetic technique in the vicinity of a major artery or vein AND one of the following: clearly identified bleeding from needle or catheter, bleeding from injured vessel documented by imaging study (eg, ultrasound, angiogram), bleeding from injured vessel diagnosed by other clinical findings (rapid hematoma formation [eg, hemothorax], pulsating hematoma), or bleeding from vessel confirmed by surgical exploration.
- Pneumothorax: regional anesthetic technique in the vicinity of the lung AND clinical diagnosis of (tension) pneumothorax and insertion of chest tube or diagnosis of pneumothorax by imaging study (eg, x-ray, CT, ultrasound).
- 11. Unintentional puncture of an adjacent organ: puncture or catheter placement in the vicinity of the affected organ (eg, kidney, liver, intestines, etc) AND visualization of needle or catheter entry in the affected organ by imaging (eg, ultrasound, CT) or structural organ damage consistent with needle entry, catheter placement, or drug injection on imaging or during surgical exploration.
- Respiratory depression/arrest: respiratory arrest/hypoventilation or distress related to regional anesthetic/analgesic procedure itself but not to procedural sedation/analgesia AND initiation of ventilatory support or hypoxia (Sao₂ <80% or Pao₂ <50 mm Hg) without supplemental oxygen.
- 13. Cardiac arrest: regional anesthetic/analgesic procedure injection performed and event related to the procedure in the judgment of the practitioner AND asystole, ventricular fibrillation, or pulseless electrical activity.