20-2-00543-21
AFS 75
Affidavit In Support
12413251

FILED
LEWIS COUNTY
2022 MAY 20 AM 11:53
SUPERIOR COURT
CLERK'S OFFICE

THE HONORABLE JAMES W.

Date of Hearing: June 1, 2022 @ 9:00 a.m.

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON, LEWIS COUNTY

SCOTT HAMILTON, as guardian ad litem for Z.H., Plaintiffs, vs. LINDA AMONDSON-MULLER, Personal Representative of the ESTATE of LAURA HAMILTON, Defendants.	NO. 20-2-00543-21 DECLARATION OF SUSAN MACHLER IN SUPPORT OF PLAINTIFF'S MOTION RE: EXPERT TESTIMONY

SUSAN MACHLER, under penalty of perjury under the laws of the State of Washington, makes the following declarations:

1. My name is Susan Machler, and I am competent to testify to matters contained herein. I am one of the attorneys of record in the above-entitled case.

2. Attached hereto as Exhibit 1 are true and correct copies of excerpts from the deposition transcript of Dolly Browder.

3. Attached hereto as Exhibit 2 are true and correct copies of excerpts from the deposition transcript of Michele Grimm, Ph.D.

4. Attached hereto as Exhibit 3 are true and correct copies of excerpts from the deposition transcript of Mark Scher, M.D.

OSBORN MACHLER
2025 First Avenue, Suite 1140
Seattle, WA 98121
206-441-4110 (Tel)
206-441-4220 (Fax)

4. Attached hereto as Exhibit 4 is a true and correct copy of *The Epidemiology of Brachial Plexus Birth Palsy in the United States: Declining Incidence and Evolving Risk Factors*.

5. Attached hereto as Exhibit 5 is a true and correct copy of *Assessment of Current Epidemiology and Risk Factors Surrounding Brachial Plexus Birth Palsy*.

6. Attached hereto as Exhibit 6 is a true and correct copy of *Anatomical Variations of Brachial Plexus in Fetal Cadavers*.

7. Attached hereto as Exhibit 7 is a true and correct copy of Dr. Raymond Tse's operative report.

8. Attached hereto as Exhibit 8 is a true and correct copy of *Brachial Plexus Variations During the Fetal Period*.

9. Attached hereto as Exhibit 9 are true and correct copies of excerpts from the deposition transcript of Eric Knowles.

Dated this 19th day of May, 2022, at Seattle, Washington.

Susan Machler

OSBORN MACHLER
2025 First Avenue, Suite 1140
Seattle, WA 98121
206-441-4110 (Tel)
206-441-4220 (Fax)

CERTIFICATE OF SERVICE

The undersigned hereby certifies under penalty of perjury under the laws of the State of Washington that I caused the foregoing to serve upon the following in the manner indicated below:

Attorneys for Defendant:

Donna Moniz
925 4th Ave, Ste. 2300
Seattle, WA 98104

☐ Via Electronic Filing
☐ Via Legal Messenger
☐ Via U.S. Mail
☒ Via E-Mail: monizd@jgkmw.com; vasquezb@jgkmw.com; randp@jgkmw.com; sproulj@jgkmw.com
☐ Via Fax:

Dated this 20th day of June, 2022 at Seattle, Washington.

Jenine Michaelis

OSBORN MACHLER
2025 First Avenue, Suite 1140
Seattle, WA 98121
206-441-4110 (Tel)
206-441-4220 (Fax)

EXHIBIT 1

SUPERIOR COURT OF WASHINGTON, LEWIS COUNTY

SCOTT HAMILTON, as guardian ad litem for Z.H., Plaintiff,	)	
vs.	)	20-2-00543-21
LINDA AMONDSON-MULLER, Personal Representative of the Estate of Laura Hamilton, Defendant.	)	

VIDEOTAPED REMOTE DEPOSITION UPON ORAL EXAMINATION OF

DOLLY BROWDER

9:00 a.m.

APRIL 13, 2022

(Via Zoom)

REPORTED BY: ELEANOR J. MITCHELL, RPR, CCR 3006

Q. Wow. They moved to the big city, sounds like?
A. Yeah, they did.
Q. How old are they right now?
A. My older daughter is 44, and my younger daughter is 41.
Q. And are those children from your marriage?
A. They are.
Q. Do you remember if you delivered them in a hospital or if you had a home birth?
A. How can I forget? Does any mother forget where she delivers. Oh, I had both of them at home.
Q. And I'm assuming that neither of them suffered a birth injury; is that right?
A. That's correct.
Q. Can you tell us about the extent of your medical education?
MS. MONIZ: Object to the form.
THE WITNESS: Is there an objection, Donna?
MS. MONIZ: Yes. That's an objection as to the term "medical education." But you may answer, if you're able to.
A. Well, I'm not sure what you mean by medical education. My degree is in speech and hearing pathology and -- from the University of Washington, and

that's my bachelor's degree. And I did have anatomy and physiology during that, which is part of most medical training programs.

And then after that, I mean, I don't know what you want me to say for my midwifery training, but that's much more extensive.

Q. Well, yeah. Let's -- do you consider midwifery training to be medical training?

A. Some of it is. Not all of it. Most of it is midwifery training, which is different than medical training in some ways.

And some of it overlaps, and some of it is different, which why I think a lot of times we either agree or disagree on certain procedures.

Q. Okay. So is there anything else to add? Any other medical training -- or any other medical education that you received?

MS. MONIZ: Object to the form.

A. I'm -- you need to be more specific about "medical training." I -- I'm not a doctor. And I'm not a nurse. I'm the midwife.

Q. Okay. So the term "medical education" is confusing?

A. It is.

MS. MONIZ: Object to the form.

Q. (BY MR. NEFF.) Okay. Well, have you ever attended medical school?

A. Medical school?

Q. (Nodding.)

A. No.

Q. Are you a registered nurse?

A. I am not.

Q. Have you ever attended any nursing school?

A. I attended some program -- or some classes that nurses attend, and a lot of the midwifery training programs have nursing standards and classes that can be taken.

Q. And so aside from those classes and that education, what about any medical training?

MS. MONIZ: Object to the form.

A. Can you be more specific about what you mean by "medical training"?

Q. (BY MR. NEFF.) Any training related to the field of medicine. Have you had any of that?

MS. MONIZ: Object to the form.

THE WITNESS: Should I go ahead and answer? I'm not quite sure how to answer.

Q. (BY MR. NEFF.) Yeah, you can answer. I'm -- we'll go ahead --

MS. MONIZ: You can answer, if you're able to. I mean, I -- I don't know if this is a semantic problem or intentionally trying to discredit the witness, but she's trying to tell you she's a midwife, and she has midwifery training.

A. Yeah. I mean, being a midwife is definitely different than being a doctor.

Q. (BY MR. NEFF.) Okay. But --

A. And --

Q. -- I mean, can we agree that midwives are in the medical field?

MS. MONIZ: No.

A. I don't see them in the standard medical field.

Q. (BY MR. NEFF.) Okay.

A. There are some midwives who are, and some who are not. And the midwives that attend out-of-hospital births deal with normal pregnancy and birth. It's not an illness; whereas, most physicians and nurses in hospitals work with people who are sick.

Q. Okay. So there are some midwives in the medical field, and some that are not. Were you in the medical field as a midwife?

A. No.

Q. Can you explain to us what it means to be a certified professional midwife?

EXHIBIT 2

SUPERIOR COURT OF WASHINGTON, LEWIS COUNTY

SCOTT HAMILTON, as guardian ad litem for Z.H.,	)	
Plaintiff,	)	
vs.	)	20-2-00543-21
LINDA AMONDSON-MULLER, Personal Representative of the ESTATE OF LAURA HAMILTON,	)	
Defendant.	)	

REMOTE DEPOSITION UPON ORAL EXAMINATION OF

MICHELE GRIMM, Ph.D.

11:00 a.m.

APRIL 14, 2022

(Via Zoom)

REPORTED BY: ELEANOR J. MITCHELL, RPR, CCR 3006

and biomedical engineering at Michigan State University.

Q. Now, other than reviewing documents, did you do any other kind of tests or analysis to form your opinions in this case?

A. I did not do any modelling of the specific delivery, so my opinion is based on the knowledge about the mechanisms of injury that I have gained from my research, along with my understanding of the literature, and then assessing how the facts of the case fit in with our current knowledge regarding causes for this particular type of injury.

Q. And in your education, do you have any medical training?

A. Not clinical medical training. I have learned biomedical science alongside engineering from freshman year all the way through my Ph.D. I did take gross anatomy with the medical students at the University of Pennsylvania, and my physiology course at Hopkins, which was a total of 12 credits over two semesters. It was taught by the same faculty who taught the medical school physiology course.

Q. All right and do you have -- so do you have any formal obstetrical training?

A. I do not.

Q. Do you have any formal neurology training?
A. No, nothing in clinical disciplines of medicine.
Q. Have you had any nursing training?
A. No.
Q. Or midwife training?
A. No.
Q. Now, let's see. Now, in -- in your professional career -- well, let's...
Have you -- you've published on the -- on the subject of shoulder dystocia and brachial plexus injuries; is that correct?
A. Yes, I have.
Q. How many published works have you -- do you have?
A. I have three papers and a peer-reviewed book chapter on brachial plexus injury and shoulder dystocia. I have a further paper talking about the effect of maternal forces during shoulder dystocia, a review article regarding the forces of labor and delivery, and then I was a co-author on this ACOG 2014 task force publication on neonatal brachial plexus palsy.
Q. And when was the last time you published anything on the subject of shoulder dystocia and/or

EXHIBIT 3

IN THE SUPERIOR COURT OF THE STATE OF WASHINGTON, LEWIS COUNTY

SCOTT HAMILTON, as guardian ad litem for Z.H., Plaintiffs,	)
vs.	) NO. 20-2-00543-21
LINDA AMONDSON-MULLER, Personal Representative of the ESTATE of LAURA HAMILTON, Defendants.	)

DEPOSITION UPON ORAL EXAMINATION

OF

MARK SCHER, M.D

Date: April 6, 2022

Location: Remote Locations via audio-visual conferencing

Start Time: 12:00 p.m.

End Time: 1:45 p.m.

Reported By: Alison J. Sosa, CCR
CCR # 2575

testify about damages; is that correct?
A Yes, that's right.
Q Were you provided with any photos of Zachary?
A No, I was not.
Q And so in terms of his functioning, what are you relying on?
A Certainly the parents' depositions themselves so I have an idea at least up to the time that they were deposed what they saw as the family. Supplemented by the treaters and the school, the IEP participants with Zachary's school care. And also whatever clinic notes and mental health clinics were available. So anything that was documented that dealt with Zachary's care.
Q Okay. All right. Okay. What did you do to prepare for this deposition?
A I looked at all the records again. I looked at parts of the depositions. Not all of them. Certainly Ms. Johnson's deposition I recently looked at. Only because I -- you should know I spoke to Ms. Penilton, the Defense Life Care Planner. Although I don't see a report that she's generated yet. And so clearly based on my discussion with Ms. Penilton. When a couple of days ago the deposition of Cloie Johnson came, I looked at it because of that recent conversation I had with the Defense Life Care Planner. And that's it. In general some literature, but not -- not exhaustively.

important based on causation for me. Not -- not based on delivery technique, but based on where the brachial plexus was relative to the injury.

Q All right. Did Laura Hamilton's records say that at the time of delivery that the baby was right occiput anterior?

A I don't recall whether she said it. I know that it was in the records.

Q It's in her records? In her delivery records?

A I don't know if they were in her records. I know they were in delivery records. They were in hospital records. Excuse me.

Q Okay. And how would the hospital know?

A Well, she would have to tell them because this was an in-home delivery.

Q All right. And then you go on to say, "Cases of brachial plexus injury when presentation is right occiput anterior, the posterior arm is caught higher in the vaginal tract on the sacral promontory."

So on what basis do you form that opinion?

A Well, as a doctor, my knowledge of where the sacral promontory is relative to the symphysis pubis, which is lower down. And my experience with children who presented with a posterior shoulder impaction that's higher up earlier during the second stage of labor. I mean, once again, I'm not an obstetrician. I'm not a midwife. I'm not trying to give posterior shoulder, it allows me to at least understand post hoc, because I can't do anything about it, how it happened.

Q Other than a statement in the medical records, is there any other evidence in this case that the baby presented right occiput anterior?

A No.

Q And it's your opinion that this child suffered avulsion -- five avulsions before shoulder dystocia even incurred --

A Yes.

Q -- on a more probable than not basis?

A Yes.

Q Based on one statement in one medical record that this child presented right occiput anterior?

A Yes. Based on all the facts that I'm -- will be discussing subsequently with you, yes.

Q Well, what other facts are there?

A Well, my knowledge -- assuming it was posterior presentation. I mean, I won't go on to answer any more because you'll be asking me that based on the disclosure.

And the position of the shoulder in which -- will increase the angle from the -- of the head and -- away from the shoulder, because the neck -- angle of the neck gets increased. It's important for me to know whether it's anterior or posterior. The posterior presentations, as I've explained to you that I've learned from the literature of

Now that I said that, one needs to extrapolate before birth, because, obviously, we can't measure tone in the womb. And so we have to approximate -- I have to approximate as a neurologist what's the baby look like at birth and then at the one-minute Apgar when tone could be assessed, and then extrapolate back, knowing that babies normally have decreased tone in order in facilitate an easier delivery.

When babies are experiencing descent with uterine contractions, there will be compression of the head. There will be some changes that will affect the heart rate. And as a result physiologically -- not pathologically -- they will become bradycardic maybe, or hypoxic a little bit. That will make them relax. That's important as an evolutionary protective mechanism to prevent injury.

So we're talking about transient, short-term hypotonia, which in normal sense is an adaptive response to parturition, to the delivery process.

For Zachary, unless I'm jumping too far ahead, Zachary had the need for mouth to mouth resuscitation by Ms. Hamilton at zero minutes. And then at one minute had a reduced score on the Apgar for tone of one out of two.

So clearly at birth and into one minute, there was some degree of tone. We don't know at birth if it's worse than at one minute. And knowing full well the shoulder dystocia and the preceding posterior arm -- arm/shoulder

ROA would ask -- would cue me in as the consultant clinically in Pittsburgh and then in Cleveland. Say to the OB or the midwife who delivered, "What was the maneuvers that you had to do in terms of where that shoulder that's -- that I'm seeing has the brachial plexus paresis, where was it -- where was the shoulder?" And that orientation of ROA is associated from my experience as a child neurologist, neonatal neurologist, to be posterior more than anterior. I have to have verifications of that by the treater, by someone that was actually there. Not me.

Q So is there -- is there verification from somebody who was actually there that the injured shoulder was posterior?

A Not that's documented in the records. Because it was a home delivery. I mean, to the best of the ability that I could tell from the records, no, there's nothing else.

Q Okay. Now I want to go back to the hypotonia. And you talked about the Apgar score. And so can you -- well, let's talk about the Apgar score.

A Sure.

Q You know, and the Apgar score is the -- actually, there's a Apgar scoring chart; isn't that correct?

A Sure. There are five items in the Apgar score, sure.

Q Okay. And the score that -- that the clinician, in this case the midwife, chooses for each five things, chooses one of three things; is that correct?

A Well, they have -- there's a point of -- there's a score of zero, one, or two. There are five items, yes.

Q Okay. So this is -- so -- so the clinician has to -- has to fit with what they're observing into what is set forth in the Apgar scoring chart; is that --

A Sure. That's fair.

Q Okay. And so in this case Laura Hamilton, she scored muscle tone as one; is that correct?

A Yes.

Q And so how -- which says some flexion of extremities. And so now much hypotonia was there?

A There's no way of quatifying. That's a good question. It's a quantitative observation that I would make too, but that Laura Hamilton made. But one cannot quantitate it. Someone can -- one can give the traction response or the angle that you get a resistance to stretch that would allow you to say that the baby was a traction response of one. And also the way the baby is sitting or moving or, let's say, laying will give you a sense of resting tone. And for that she scored a one out of two.

Now, this is also the same individual, Laura Hamilton, who had to give mouth to mouth to Zachary at birth. If you have to give mouth to mouth to a baby at birth, I can't imagine the Apgar score was even one, the tone is one. Could be even less. But clearly it was reduced at one minute when of life."

Q Okay. So I have another question. I'll ask it.

So but in your disclosure, though, it says, "Dr. Scher will explain that an excessive degree of lower tone resulted in excessive stretching of Z.H.'s shoulder girdle."

A Yes.

Q And so from -- from an Apgar score of one, which is not a zero, how do you come to the conclusion that the hypotonia that he experienced was excessive?

MS. MONIZ: Object to the form.

A Well, he -- he -- by one minute he was still reduced in tone. And if one walks that back in time not just to zero minutes at birth, but even before, knowing that there's a physiologic hypotonia, that protective reflex was working against him causing excessive stretch beyond elasticity of his tissues around his brachial plexus causing injury.

Q Okay. And do you have any idea how Laura Hamilton evaluated Zachary's muscle tone at one minute?

A Other than my general knowledge of how nurses and physicians are trained to look -- as I mentioned earlier, traction responses, you move the limb around the joint until you feel the tug of the child against that movement. That would be a traction response. That would be one way she could have -- she could measure it.

The other is by looking at the actual resting posture

Q Okay. And if she was looking at his -- his resting posture, that -- that, you know, the -- the right arm was -- would it appear - Well, I'm going to ask you. Not tell you. Would the right arm appear flaccid or weak or something? Would there be -- would she be able to observe an issue with that right arm when she was observing his resting posture?

MS. MONIZ: Object to the form. Object to the form of the question.

A So I'm not there at the time. So contemporaneously there's a lot of different factors that she as the midwife delivering a baby at home is dealing with in terms of safety of the mother, safety of the baby. She obviously and she most likely after the fact put together her memory of what she saw to score the Apgar. I'm sure she didn't do it at the time. She was busy taking care of mother and baby.

Whether there was something observable that she did not record that she could have recorded, I have no idea because it's not in the records. I have no idea how to answer your question. All I can tell you is the resting tone can give you a score of one out of two. And your traction response can give you a score of at least one out of two, and maybe perhaps segmentally on the right -- on the affected arm, zero out of two. I don't know. But it's not documented in the records for me to know that.

Q Okay. And so is also -- is it also -- it's also true that

you can't know whether the rest of Zachary was actually a two outside -- if it -- outside of the injured right arm?
A No.
MS. MONIZ: Object to the form of the question.
A That would not be my experience with the Apgar score as I've observed through the years they're done. The Apgar score is considered a global score of tone. Global meaning axial tone and appendicular tone. So it's looking at a global score of the baby. Usually resting tone is what you look at and you give the score.
If -- if the examiner wants to go through the range of motion, as I said, the traction response, that's added onto it. But if you look at any Ballard score or any Dubowitz score, they don't even include traction response. They just say, "What's the position of the arms; what's the position of the legs based on angle, of the popliteal angle, and of the -- of the elbow angle?" And they give a score. And so, no, I -- I disagree with your -- your question in saying that this is a global score. So the overall score was one out of two.
Q All right. And so if Laura Hamilton had made observations, you know -- you don't know what -- you haven't reviewed her deposition, have you?
A No.
Q So if she made -- if she described observations that she made

about the baby, you don't know what those are?

A Well --

MS. MONIZ: Object to the form.

A -- having -- not reading -- having not read her dep, that would be the only testimony I would have available to me. But, no, I haven't read her deposition.

Q Okay. And in the literature that you talk about, do the studies -- are there studies that you included in the disclosure that -- that talk about the association of hypotonia with complete brachial plexus avulsion?

A I believe I shared with the attorney I worked with writing the disclosure at least two papers that control for in a multi-varied analysis of muscle tone as a variable that's associated with brachial plexus injury. Yes. DeFrancesco was one and I'm blocking on the other. Abzug was the other article.

So these are recent articles. 2018, I believe. Because these require large enough data sets and the right statistical analysis to control for other variables. But they confirm what I've learned through the years. I've known about the risk of hypotonia since my medical school days. But those are the most recent pieces of literature that I can share with you; Abzug and DeFrancesco.

Q Okay. I see those on here. Okay. And so if -- so do these studies tell us the incidence of brachial plexus injury

occurring along with hypotonia?
A Those particular articles among other variables that they were able to control for given the statistical limits of their population size, their sample size, yes. They were able to control for the other variables and say, "Aha, hypotonia is a variable to consider."
I'm not here to tell you it's the only variable. There are multiple variables that could be in play.
Q Okay. So I want to talk about the anatomical variations of the brachial plexus. So is it your opinion that -- that Zachary and his mother had anatomical variations?
A In essence, yes.
Q Okay. And what anatomical variations did they have?
A To the limits of what I can see in the records, I don't know. The fetal surveillance before birth and the postnatal studies, which include the MRI of Zachary when they were preparing to do his grafting, et cetera, are not sensitive and specific enough to tell you exactly. And postinjury the scarring would not allow you to see it. These are very subtle.
But one thing about biological variations, we as humans need to have biological variations as primarily a protective series of mechanisms to prevent injury. But the unfortunate situation which can't be predicted based on the records, is that the anatomical variation of mother and of

the baby contributed to the injury of the posterior -- of the shoulder with posterior presentation.

Q Okay. So you indicated that -- strike that.

So is there a way for you to determine -- like, to go backwards in time after the fact and postulate what kind of anatomical variations existed that contributed to this injury?

MS. MONIZ: Object to the form of the question.

A So the answer is based to the level of what's documented, no. I think I mentioned that earlier. The surveillance and the postnatal testing would not allow you to do that because of the testing and because of the scarring around the brachial plexus. It's my knowledge and experience as a neurologist I many times will see in a patient with any injury to any part of the nervous system my knowledge of developmental neuroanatomy allows me to make a certain conclusion based on my training and experience.

Q Okay. And in the list of literature that you provided us, are there studies that associate or discuss anatomical variations with brachial plexus avulsion injuries?

A Yes. The two -- the two main ones are the Turkish studies by Kirik, K-I-R-I-K, and Wozniak, the Polish researcher. Those two groups in those two countries had spent the greatest amount of time with cadaveric, meaning post death and postmortem evaluations to document as much as 50 percent

EXHIBIT 4

The Epidemiology of Brachial Plexus Birth Palsy in the United States: Declining Incidence and Evolving Risk Factors

Christopher J. DeFrancesco, BS, Divya K. Shah, MD, MME,† Benjamin H. Rogers, BA,* and Apurva S. Shah, MD, MBA**

Background: The epidemiology of brachial plexus birth palsy (BPBP) in the United States may be changing over time due to population-level changes in obstetric care.

Methods: The Kids' Inpatient Database from 1997 to 2012 was analyzed. Annual estimates of BPBP incidence and disease determinant distribution were calculated for the general population and the study population with BPBP. Long-term trends were analyzed. A multivariate logistic regression model was used to quantify the risk associated with each determinant.

Results: The database yielded a combined total of 5,564,628 sample births extrapolated to 23,385,597 population births. The population incidence of BPBP dropped 47.1% over the 16-year study period, from 1.7 to 0.9 cases per 1000 live births ($P<0.001$). Female, black, and Hispanic subgroups had moderately increased risks of BPBP. Among children with BPBP, 55.0% had no identifiable risk factor. Shoulder dystocia was the strongest risk factor for BPBP in the regression model [odds ratio (OR), 113.2; $P<0.001$], although the risk of sustaining a BPBP in the setting of shoulder dystocia decreased from 10.7% in 1997 to 8.3% in 2012 ($P=0.006$). Birth hypoxia was independently associated with BPBP (OR, 3.1; $P<0.001$). Cesarean delivery (OR, 0.16; $P<0.001$) and multiple gestation birth (OR, 0.45; $P<0.001$) were associated with lower incidence of BPBP. Notably, the rate of cesarean delivery increased by 62.8% during the study period, from 20.9% in 1997 to 34.0% in 2012 ($P<0.001$).

Conclusions: Over a 16-year period, the incidence of BPBP fell dramatically, paralleled by a significant increase in the rate of cesarean delivery. Systemic changes in obstetric practice may have contributed to these trends. As more than half of BPBP cases have no identifiable risk factor, prospective investigation of established risk factors and characterization of new disease determinants are needed to more reliably identify infants at greatest risk. Racial and geographic inequalities in disease burden should be investigated to identify interventional targets.

Level of Evidence: Level III—case series.

From the *Division of Orthopaedics, Children's Hospital of Philadelphia; and †Department of Obstetrics and Gynecology, University of Pennsylvania, Philadelphia, PA.

None of the authors received financial support for this study.
The authors declare no conflicts of interest.

Reprints: Apurva S. Shah, MD, MBA, Division of Orthopaedics, The Children's Hospital of Philadelphia, 3401 Civic Center Boulevard, Philadelphia, PA 19104. E-mail: shaha6@email.chop.edu.

DOI: 10.1097/BPO.0000000000001089

Key Words: brachial plexus palsy, birth injury, shoulder dystocia, cesarean, obstetric

(*J Pediatr Orthop* 2019;39:e134–e140)

Brachial plexus birth palsy (BPBP) is defined as flaccid weakness of the upper limb that occurs as a result of traction to the brachial plexus during birth. Disease incidence has previously been estimated between 0.4 and 5.1 per 1000 live births.[1–4] Spontaneous neurological recovery occurs in 66% to 92% of cases,[1–4] but microsurgical treatment consisting of nerve transfers and/or nerve grafting may be indicated in infants with persistent motor deficits.[2,5–7] Children that do not spontaneously recover antigravity elbow flexion by 3 to 6 months of age will have permanent neurological impairment, with high associated financial and societal burdens.[2] As nearly half of affected families pursue litigation, this condition is also responsible for a high legal burden.[8,9] Further, families that pursue malpractice cases commonly feel undereducated about the injury and carry undue psychological burdens—including high rates of depressive symptoms and anger.[9] Such issues make the epidemiology of BPBP an important topic for obstetricians and the orthopaedists that subsequently care for and counsel affected families.

Unfortunately, a thorough understanding of the epidemiology of BPBP and its risk factors remains lacking. Estimates of disease incidence from institutional and regional studies are difficult to generalize nationally.[2,4] In addition, previous investigations have suggested that the incidence of BPBP was rising before 1997[10] and remained steady thereafter,[10] whereas other work has indicated that disease incidence dropped after 1997.[1] Although a multitude of risk factors for BPBP have been described, it remains difficult to identify which infants are at greatest risk, indicating a need for improved characterization of disease determinants.[11–17]

Recognizing these issues, Foad et al[1] used a large national database of inpatient pediatric hospital stays—the Kids' Inpatient Database (KID)—to examine the national epidemiology of BPBP. Using data sets from 1997 to 2003, the investigators found a cumulative incidence of 1.5 cases per 1000 live births. Although the study offered an informative analysis, the assessment spanned only 7 years, making it difficult to draw conclusions regarding long-term temporal trends in disease incidence.

The main purpose of this study was to use more recent data from the KID to provide an updated report on the incidence of BPBP, the distribution of associated factors, and major trends in these parameters over a 16-year period.

METHODS

Database

The KID is an administrative database released at 3-year intervals by the United States Healthcare Cost and Utilization Project (HCUP).[18] Each release constitutes a 1-year sample of pediatric discharges from all non-rehabilitation, nonfederal, short-term public hospitals in states participating in HCUP. Discharges are assigned to 1 of 3 categories: complicated births, uncomplicated births, or other pediatric hospital stays. Only live born infants are included as birth observations. Approximately 10% of all uncomplicated births and 80% of complicated births are sampled. The KID provides a sampling weight for each discharge based upon hospital characteristics, thereby permitting extrapolation of sample findings to population-level estimates. Each KID release includes a core file with patient variables such as age, sex, International Classification of Diseases 9 (ICD-9) diagnosis and procedure codes, and length of stay. Released along with each core file is a hospital file, which includes institution-level information like hospital size (based upon bed number), teaching status, location (rural or urban), and geographic region. The data for this study were drawn from the core and hospital files from all available KID releases: 1997, 2000, 2003, 2006, 2009, and 2012.

Defining Variables

All variables were defined using ICD-9 codes. BPBP (ICD-9: 767.6) was the primary outcome variable. Exposure variables (hereafter referred to as "disease determinants") included shoulder dystocia (ICD-9: 763.1), fetal macrosomia >4.5 kg (ICD-9: 766.0), other heavy-for-dates infants (ICD-9: 766.1), breech delivery (ICD-9: 763.0), instrumented birth with forceps (ICD-9: 763.2) or vacuum (ICD-9: 763.3) assist, cesarean delivery (ICD-9: 763.4, V30.01, V31.01, V33.01, V34.01, V37.01, V39.01), and multiple gestation with ≥2 fetuses (ICD-9: V31.00, V31.01, V32.00, V32.01, V33.00, V33.01, V34.00, V34.01, V35.00, V35.01, V36.00, V36.01, V37.00, V37.01). It was hypothesized that fetal hypoxia increases the risk of BPBP. Birth hypoxia was defined using ICD-9 codes (768.5, 768.6, 768.70, 768.71, 768.72, 768.73, 768.9, 770.88) as well as Clinical Classification Software Diagnosis (DXCCS) code 220. Birth hypoxia is not reported in this study for the year 1997 because DXCCS codes were not consistently reported before the year 2000.

Statistical Analysis

Weighted counts were used to yield population-level estimates of disease incidence and the frequency of disease determinants. Accompanying standard errors and confidence intervals (CIs) were calculated using first-order Taylor series linear approximations. CIs around frequency and proportion estimates were calculated using logistic transforms. Adjusted Wald tests were used to check for differences between 1997 and 2012 estimates. A type I error rate of 0.05 defined critical *P*-values for statistical significance. Stata 14.2 (StataCorp.; College Station, TX) was used for all statistical calculations in this study.

Population frequencies of newborn-based demographic characteristics including sex, race, and payer status were first estimated. Next, a frequency estimate for each disease determinant was calculated as the proportion of all live births with the specified diagnosis. This estimation process was repeated in the subgroup of cases of BPBP rather than all births. Rates of BPBP were also computed for population subgroups defined by each disease determinant. Determinant-specific unadjusted relative risk was estimated by comparing disease rates between subgroups with and without each disease determinant.

A multivariate logistic regression model was fit to the data. Birth hypoxia was included after univariate logistic regression suggested a relationship with BPBP ($P<0.001$). Testing of exposure variables did not reveal a high degree of collinearity among included inputs. Appropriate interaction terms were included in the model. Demographic variables including patient-based characteristics (sex, race, and payer status) and hospital-based characteristics (hospital size, location, teaching status, and region) were added to the model to account for any related effects.

RESULTS

The database yielded 5,564,628 sample births extrapolated to 23,385,597 live births in the population (95% CI, 22,780,168-23,991,026). Patient demographics changed during the study period, with increasing proportions of patients with public insurance or minority race. Demographic information is summarized in Table 1. The overall rate of BPBP during the study period was 1.2 per 1000 births. However, this rate dropped steadily from 1.7 per 1000 births in 1997 to 0.9 per 1000 births in 2012 ($P<0.001$), representing a 47.1% change over 16 years (Fig. 1).

Table 2 summarizes the yearly distribution of disease determinants among all live births without regard to BPBP (the population at-large), showing that rates of macrosomia, instrumented delivery, and birth hypoxia decreased significantly over the study period. The baseline incidence of macrosomia decreased by 56.9% between 1997 and 2012 [0.56% in 1997 vs. 0.24% in 2012; $P<0.001$ (Table 2 and Fig. 1)]. Simultaneously, the rate of cesarean delivery rose by 62.8% [20.9% in 1997 vs. 34.0% in 2012; $P<0.001$ (Table 2 and Fig. 1)]. Rates of heavy-for-dates infants and multiple gestations increased modestly.

Table 3 details information for the subgroup of patients with confirmed BPBP. This shows that the incidence of shoulder dystocia, macrosomia, heavy-for-dates infants, breech delivery, instrumented birth, and hypoxia (risk factors) was higher among BPBP patients compared with the population at-large. In contrast, cesarean delivery and multiple gestation (protective factors) were less common. It is also notable that 55.0% of cases of BPBP were not associated with any of the investigated risk factors.

TABLE 1. Demographics of Study Population, 1997 to 2012

	1997	2000	2003	2006	2009	2012	All Years
Total live births	3,747,145	3,887,891	3,920,787	4,106,487	3,989,527	3,733,760	23,385,597
Female (%)	48.81	49.01	48.75	48.85	48.85	48.91	48.86
Race (%)							
White	61.12	57.87	52.94	51.68	51.97	52.39	54.60
Black	15.20	13.02	12.62	13.00	13.85	14.32	13.65
Hispanic	16.04	19.85	23.72	24.68	22.94	20.05	21.29
Asian/Pacific Islander	3.43	3.73	4.22	4.42	4.65	5.54	4.33
Native American	0.50	0.46	0.47	0.59	0.90	0.89	0.64
Other	3.71	5.07	6.03	5.63	5.69	6.82	5.49
Payer (%)							
Private Insurance	56.57	57.93	53.44	49.98	47.41	46.18	51.89
Medicaid	34.13	34.35	38.70	42.13	45.50	46.37	40.23
Other	9.30	7.72	7.87	7.88	7.10	7.45	7.88

Table 4 describes the unadjusted incidence of BPBP based on the presence or absence of each disease determinant. Although infants without shoulder dystocia had a 0.10% rate of BPBP, those with shoulder dystocia had a 9.9% rate, making shoulder dystocia the strongest risk factor for BPBP. The risk of sustaining a BPBP in the setting of shoulder dystocia decreased from 10.7% in 1997 to 8.3% in 2012 ($P=0.006$). Figure 2 shows that the incidence of BPBP rises significantly with the number of risk factors, reaching 17.9% in infants with ≥3 identified risk factors—145 times greater than the population risk. Of note, only 6.5% of all live births had any identified risk factor for BPBP and <0.2% had ≥2 risk factors.

A goodness of fit test[19] revealed good agreement between the multivariate logistic regression model and the population-level data (fit could not be rejected; $P=0.131$). The model confirmed shoulder dystocia as the greatest identified risk factor for BPBP, with an odds ratio (OR) of 113.2 (95% CI, 104.9-122.2, Table 5). Birth hypoxia was independently associated with an approximately 3-fold increase in the risk of BPBP (OR, 3.08; 95% CI, 2.60-3.64). This association is similar in magnitude to that found for

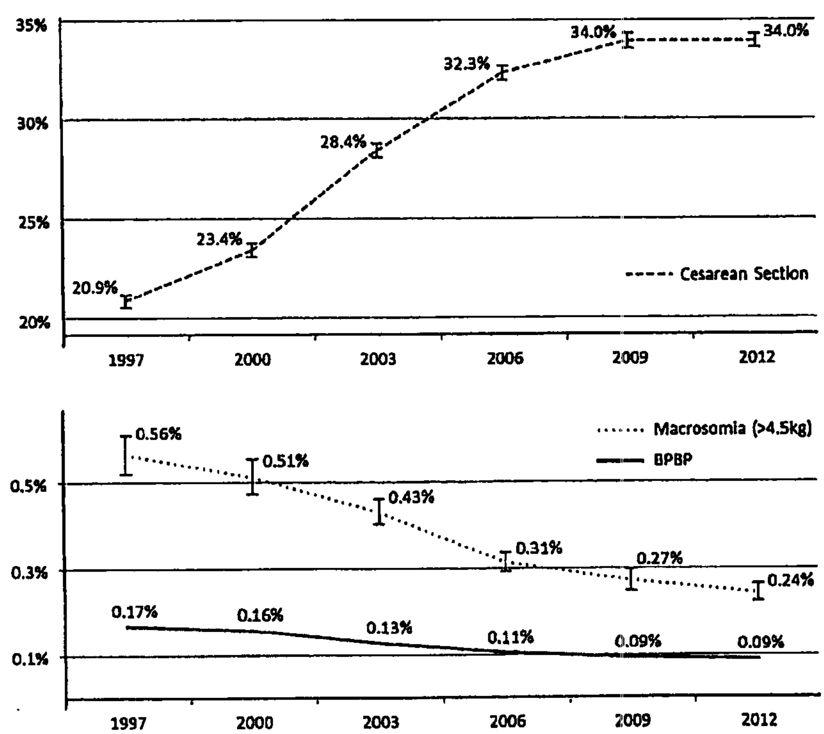


FIGURE 1. These graphs show the estimated incidence of BPBP as well as the yearly prevalence for 1 major risk factor (macrosomia) and 1 major protective factor (cesarean delivery). Note the different sets of axes. The error bars illustrate 95% confidence intervals. These intervals were not included with BPBP incidence due to their small size. The rate of BPBP dropped by nearly half while rates of cesarean delivery increased by 62.8% over the same period. BPBP indicates brachial plexus birth palsy.

TABLE 2. Estimated Prevalence of Disease Determinants for Brachial Plexus Birth Palsy Among US Live Births, 1997 to 2012

Variables	1997	2000	2003	2006	2009	2012	All Years	P (1997 vs. 2012)
Risk factors (%)								
Shoulder dystocia	0.27	0.24	0.20	0.19	0.23	0.26	0.23	0.6029
Macrosomia (>4.5 kg)	0.56	0.51	0.43	0.31	0.27	0.24	0.39	<0.0001
Heavy-for-dates	4.95	4.99	5.19	5.06	5.21	5.50	5.15	0.0067
Breech delivery	0.14	0.13	0.09	0.11	0.13	0.16	0.13	0.1698
Instrumented birth	0.54	0.36	0.28	0.23	0.29	0.34	0.34	<0.0001
Forceps	0.21	0.10	0.08	0.04	0.05	0.06	0.09	<0.0001
Vacuum	0.34	0.26	0.20	0.19	0.24	0.29	0.25	0.1245
Birth hypoxia	NA*	0.51	0.48	0.44	0.39	0.42	0.45	0.0046*
Protective factors (%)								
Multiple gestation	2.70	2.99	3.22	3.28	3.34	3.34	3.15	<0.0001
Cesarean	20.9	23.4	28.4	32.3	34.0	34.0	28.9	<0.0001

The corresponding *P*-value compares year 2000 data.
*The variable birth hypoxia was not available in 1997.
NA indicates not available.

breech delivery or instrumented birth. Female sex (OR, 1.27; 95% CI, 1.22-1.31), black race (OR, 1.88; 95% CI, 1.73-2.04), and Hispanic race (OR, 1.35; 95% CI, 1.27-1.44) were independently associated with moderate but statistically significant risk increases. Rates of macrosomia and heavy-for-dates were lower in infants of black or Hispanic race compared with newborns of white race (macrosomia, 0.32% vs. 0.46%; $P<0.001$; heavy-for-dates, 4.8% vs. 5.5%; $P<0.001$), whereas risks for shoulder dystocia were comparable between these groups (0.24% vs. 0.23%; $P=0.087$).

DISCUSSION

The incidence of BPBP fell steadily between 1997 and 2012, accompanied by an increase in the rate of cesarean delivery. Several national trends likely contributed to these developments. The American Congress of Obstetricians and Gynecologists (ACOG) practice recommendations on management of shoulder dystocia were revised between 1997 and 2002, stating that planned cesarean delivery to prevent shoulder dystocia may be considered for estimated fetal weights exceeding 5000 g in women without diabetes and 4500 g in women with diabetes.[20,21] The present study reveals a decrease in shoulder dystocia rates during the 4 years following these recommendations. Records also confirm that rates of primary and repeat cesarean delivery began to rise steeply around this time,[22,23] accompanied by a corresponding decrease in rates of vaginal birth after cesarean.[22] Increased use of cesarean delivery may diminish the incidence of BPBP by reducing the risk of shoulder dystocia and traction injury associated with vaginal delivery. Cesarean delivery may also indirectly lessen incidence of BPBP by facilitating delivery at an earlier gestational age and respectively lower birthweight—consistent with the downward trend in macrosomia observed during the study period.

Although higher rates of cesarean delivery likely account in large part for the change in BPBP incidence, a causal relationship cannot be confirmed by this retrospective analysis. Other changes in obstetric practice or training likely contributed, as well. For example, shoulder dystocia training courses involving simulation have been

TABLE 3. Proportion of Infants With Disease Determinants Among Those Diagnosed With Brachial Plexus Birth Palsy in the US Population, 1997 to 2012

Variables	1997	2000	2003	2006	2009	2012	All Years	P (1997 vs. 2012)
Risk factors (%)								
Shoulder dystocia	17.39	15.81	18.50	18.45	21.86	24.35	18.78	<0.0001
Macrosomia (>4.5 kg)	7.09	6.36	5.90	3.90	3.81	2.65	5.31	<0.0001
Heavy-for-dates	23.70	23.51	27.12	26.81	27.87	27.59	25.71	0.0072
Breech delivery	0.25	0.14	0.11	0.10	0.11	0.24	0.16	0.9181
Instrumented birth	3.78	2.94	2.03	1.98	2.52	2.85	2.75	0.1003
Forceps	1.51	0.88	0.83	0.63	0.65	0.55	0.91	0.0022
Vacuum	2.35	2.19	1.20	1.34	1.90	2.30	1.90	0.9020
Birth hypoxia	NA*	2.04	2.38	1.78	1.84	1.89	1.57	0.6949*
No risk factors	55.9	56.8	54.0	56.5	52.7	51.6	55.0	0.0132
Protective factors (%)								
Multiple gestation	0.92	0.82	0.66	0.51	0.76	0.38	0.71	0.0226
Cesarean delivery	2.80	3.78	4.23	5.19	6.13	6.06	4.42	<0.0001

The corresponding *P*-value compares year 2000 data.
*The variable birth hypoxia was not available in 1997.
NA indicates not available.

Copyright © 2017 Wolters Kluwer Health, Inc. All rights reserved.

TABLE 4. Unadjusted Comparison of BPBP Incidence by Disease Determinants, 1997 to 2012

Variables	BPBP Incidence 1997	BPBP Incidence 2012	BPBP Incidence All Years	P (1997 vs. 2012)	Unadjusted Hazard Ratio All Years
Risk factors (%)					
Shoulder dystocia					
Yes	10.72	8.25	9.94	0.0060	99.26
No	0.14	0.07	0.10	<0.0001	—
Macrosomia (>4.5 kg)					
Yes	2.11	0.97	1.68	<0.0001	14.42
No	0.16	0.09	0.12	<0.0001	—
Heavy-for-dates					
Yes	0.80	0.44	0.61	<0.0001	6.38
No	0.13	0.07	0.10	<0.0001	—
Breech delivery					
Yes	0.30	0.13	0.15	0.1897	1.25
No	0.17	0.09	0.12	<0.0001	—
Instrumented delivery					
Yes	1.17	0.73	1.01	0.0072	8.39
No	0.16	0.09	0.12	<0.0001	—
Forceps					
Yes	1.21	0.87	1.27	0.2971	10.38
No	0.17	0.09	0.12	<0.0001	—
Vacuum					
Yes	1.17	0.70	0.93	0.0156	7.69
No	0.16	0.09	0.12	<0.0001	—
Birth hypoxia					
Yes	0.63*	0.40	0.52	0.00371*	4.24
No	0.16*	0.09	0.12	<0.0001*	—
Protective factors (%)					
Multiple gestation					
Yes	0.06	0.01	0.03	0.0004	0.22
No	0.17	0.09	0.13	<0.0001	—
Cesarean delivery					
Yes	0.02	0.02	0.02	0.0497	0.11
No	0.21	0.13	0.17	<0.0001	—

Year 2000 data were used for these comparisons.
*1997 data were not available for birth hypoxia.
BPBP indicates brachial plexus birth palsy.

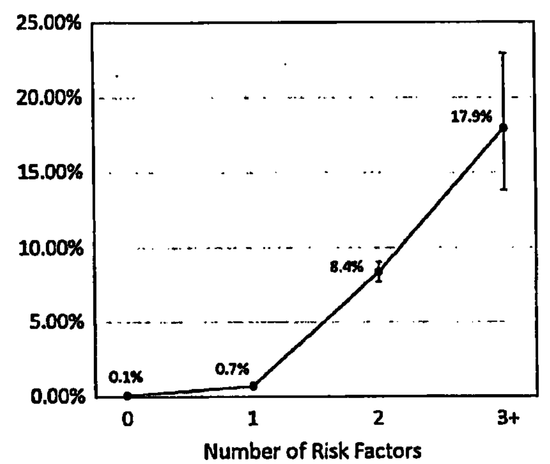


FIGURE 2. This graph illustrates the risk of BPBP with an increasing number of risk factors (protective factors excluded). The risk of BPBP shows a dose-response type relationship with the number of risk factors, rising to over 17% for those with ≥3 risk factors. BPBP indicates brachial plexus birth palsy.

shown to decrease the incidence of BPBP by a factor of 3 to 4 where implemented.[14,24] We found that the incidence of BPBP in the setting of shoulder dystocia has decreased significantly since 1997, indicating that provider response to this critical situation has improved, potentially driven by simulation-based training.

Although birth hypoxia may theoretically be confounded with other risk factors in bivariable analysis, our logistic regression accounted for interactions between hypoxia and variables like shoulder dystocia and macrosomia, showing that birth hypoxia increased the risk of BPBP independent of these other determinants. On the basis of this finding, we theorize that fetal hypotonia associated with prepartum or intrapartum hypoxia is the main mechanism for the observed relationship. Because of decreased muscle tone and dampened reflex arcs, affected infants may not be able to limit shoulder displacement as they transit the birth canal, possibly leading to increased BPBP susceptibility. To evaluate this theory, future investigators may examine relationships between BPBP and indicators of fetal or neonatal vigor that were not available in the KID, such as fetal heart tracings, cord blood gases, newborn Apgar scores, or intensive care unit admission.

TABLE 5. Multivariate Analysis of Brachial Plexus Birth Palsy Risk by Disease Determinants and Demographic Variables, 1997 to 2012

	Odds Ratio (95% Confidence Interval)
Main variables	
Risk factors	
Shoulder dystocia	113.2 (104.9-122.2)
Heavy-for-dates	8.22 (7.82-8.66)
Macrosomia (> 4.5 kg)	26.8 (24.0-30.0)
Breech delivery	3.56 (2.11-6.03)
Instrumented birth (forceps- or vacuum-assisted)	3.05 (2.56-3.65)
Birth hypoxia	3.08 (2.60-3.64)
Protective factors	
Multiple gestation	0.45 (0.32-0.63)
Cesarean delivery	0.16 (0.15-0.18)
Demographic variables	
Hospital characteristics	
Teaching hospital	1.12 (1.06-1.20)
Patient characteristics	
Female sex	1.27 (1.22-1.31)
Black race	1.88 (1.73-2.04)
Hispanic race	1.35 (1.27-1.44)
Region	
Midwest US (States: OH, MI, IN, IL, WI, MN, IA, MO, KS, NE, SD, ND)	0.88 (0.79-0.97)
West US (States: WA, MT, WY, CO, NM, AZ, UT, NV, CA, OR, HI, AK)	0.77 (0.70-0.84)

AK indicates Alaska; AZ, Arizona; CA, California; CO, Colorado; HI, Hawai; IA, Iowa; IL, Illinois; IN, Indiana; KS, Kansas; MI, Michigan; MN, Minnesota; MO, Missouri; MT, Montana; ND, North Dakota; NE, Nebraska; NM, New Mexico; NV, Nevada; OH, Ohio; OR, Oregon; SD, South Dakota; WA, Washington; WI, Wisconsin; WY, Wyoming; UT, Utah.

Several important variables could not be modeled in this analysis due to lack of data availability. Perhaps most notably, labor induction—which has been advocated in cases of macrosomia to avoid shoulder dystocia, neonatal fracture, and BPBP[25,26]—was not available. United States Centers for Disease Control and Prevention (CDC) reports indicate that labor induction rates doubled between 1990 and 2010, reaching 23.8% in 2010.[27] Increasing rates of labor induction likely contributed to the dramatic decrease in BPBP incidence observed, in part due to earlier delivery at lower birthweights. Birthweight itself was not available as a continuous variable in the KID, limiting its evaluation as a risk factor. The KID also does not record potentially important variables like maternal parity, maternal diabetes (preexisting or gestational), previous child with BPBP or shoulder dystocia, gestational age at delivery, duration of labor,[15,17] delivering provider (obstetrician vs. family practitioner vs. midwife), and unplanned (vs. scheduled) cesarean delivery.[25,28]

Our finding that over half of cases of BPBP were not associated with an identifiable risk factor is consistent with findings from prior investigations.[1,17,29] It further highlights the limitations of current models in predicting BPBP occurrence and the need for a more thorough understanding of disease determinants. Future investigations should therefore focus on identifying undescribed factors affecting the likelihood of injury. Perhaps noninvasive imaging techniques can be developed to assess the relationship between infant shoulder breadth and maternal pelvic dimensions to better identify pregnancies at high risk for shoulder dystocia and BPBP. This study also suggests that female and certain minority infants carry an increased risk of disease. Although it could be theorized that race-based inequities like this only reflect a differential distribution of risk factors, we actually found that black or Hispanic patients have lower rates of macrosomia and similar rates of shoulder dystocia when compared with white newborns. This suggests that true demographic-based disparities exist in access to adequate health care and, in particular, high-quality perinatal care. A dedicated investigation of differences in health care access and disease burden may inform public policy related to perinatal care in at-risk subpopulations.

The size of the extrapolated population of live births in this investigation is consistent with CDC records,[22] and our estimates of BPBP incidence are comparable with previous estimates.[1,29] These findings provide some validation of our analysis. In addition, our calculated 1997 to 2003 estimates matched those previously published by Foad et al.[1] As with any study utilizing a large administrative database, one limitation of this study is the potential misclassification of observations. It is also possible that the observed decline in the incidence of BPBP was not due to a true drop in disease incidence, but rather reflects increased rates of delayed diagnosis, changes in coding practice, and/or provider failure in recognizing less severe injuries. Despite these inherent limitations, this study has several strengths, including a high level of statistical power due to the KID's size.

In conclusion, this study provides a powerful analysis of the epidemiology of BPBP, revealing a marked decline in disease incidence between 1997 and 2012. Changes in the rates of cesarean delivery and alterations in obstetric training and management are likely to have played a significant role in this decline. Unidentified disease determinants undoubtedly influence disease incidence; such relationships warrant further investigation to identify targets for intervention. Because of the high morbidity associated with BPBP, improved prevention efforts are of paramount importance.

ACKNOWLEDGMENTS

The authors would like to acknowledge Divya Talwar, MPH, PhD(c), who reviewed the statistical methods of this paper for accuracy.

REFERENCES

1. Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the United States. *J Bone Joint Surg Am.* 2008;90:1258–1264.
2. Buterbaugh KL, Shah AS. The natural history and management of brachial plexus birth palsy. *Curr Rev Musculoskelet Med.* 2016;9:418–426.
3. Foad SL, Mehlman CT, Foad MB, et al. Prognosis following neonatal brachial plexus palsy: an evidence-based review. *J Child Orthop.* 2009;3:459–463.
4. Hale HB, Bae DS, Waters PM. Current concepts in the management of brachial plexus birth palsy. *J Hand Surg Am.* 2010;35:322–331.

5. Waters PM. Obstetric brachial plexus injuries: evaluation and management. *J Am Acad Orthop Surg*. 1997;5:205–214.
6. Abzug JM, Kozin SH. Evaluation and management of brachial plexus birth palsy. *Orthop Clin North Am*. 2014;45:225–232.
7. Sibbel SE, Bauer AS, James MA. Late reconstruction of brachial plexus birth palsy. *J Pediatr Orthop*. 2014;34 (suppl 1):S57–S62.
8. Chauhan SP, Chang KW, Ankumah NE, et al. Neonatal brachial plexus palsy: obstetric factors associated with litigation. *J Matern Fetal Neonatal Med*. 2017;30:2428–2432.
9. Domino J, McGovern C, Chang KW, et al. Lack of physician-patient communication as a key factor associated with malpractice litigation in neonatal brachial plexus palsy. *J Neurosurg Pediatr*. 2014;13:238–242.
10. Walsh JM, Kandamany N, Ni Shuibhne N, et al. Neonatal brachial plexus injury: comparison of incidence and antecedents between 2 decades. *Am J Obstet Gynecol*. 2011;204:324 e1–324 e6.
11. Ouzounian JG, Korst LM, Miller DA, et al. Brachial plexus palsy and shoulder dystocia: obstetric risk factors remain elusive. *Am J Perinatol*. 2013;30:303–307.
12. Hammad IA, Chauhan SP, Gherman RB, et al. Neonatal brachial plexus palsy with vaginal birth after cesarean delivery: a case-control study. *Am J Obstet Gynecol*. 2013;208:229.e1–229.e5.
13. Lindqvist PG, Ajne G, Cooray C, et al. Identification of pregnancies at increased risk of brachial plexus birth palsy—the construction of a weighted risk score. *J Matern Fetal Neonatal Med*. 2014;27:252–256.
14. Inglis SR, Feier N, Chetiyaar JB, et al. Effects of shoulder dystocia training on the incidence of brachial plexus injury. *Am J Obstet Gynecol*. 2011;204:322.e1–322.e6.
15. Grobman WA, Bailit J, Lai Y, et al. Association of the duration of active pushing with obstetric outcomes. *Obstet Gynecol*. 2016;127: 667–673.
16. Wilson TJ, Chang KW, Chauhan SP, et al. Peripartum and neonatal factors associated with the persistence of neonatal brachial plexus palsy at 1 year: a review of 382 cases. *J Neurosurg Pediatr*. 2016;17:618–624.
17. Backe B, Magnussen EB, Johansen OJ, et al. Obstetric brachial plexus palsy: a birth injury not explained by the known risk factors. *Acta Obstet Gynecol Scand*. 2008;87:1027–1032.
18. Healthcare Cost and Utilization Project (HCUP) Kids' Inpatient Database (KID). In: (HCUP) HCaUP, ed. Agency for Healthcare Research and Quality, Rockville, MD; 1997, 2000, 2003, 2006, 2009, and 2012.
19. Archer KJ, Lemeshow S. Goodness-of-fit test for a logistic regression model fitted using survey sample data. *Stata J*. 2006;6:97–105.
20. American Congress of Obstetricians and Gynecologists (ACOG) Practice Patterns. Shoulder dystocia. Number 7, October 1997. American College of Obstetricians and Gynecologists. *Int J Gynaecol Obstet*. 1998;60:306–313.
21. Sokol RJ, Blackwell SC. Bulletins-Gynecology. ACOG practice bulletin: shoulder dystocia. Number 40, November 2002. (Replaces practice pattern number 7, October 1997). *Int J Gynaecol Obstet*. 2003;80:87–92.
22. Martin JA, Hamilton BE, Ventura SJ, et al. Births: final data for 2011. *Natl Vital Stat Rep*. 2013;62:1–69. 72.
23. Barber EL, Lundsberg LS, Belanger K, et al. Indications contributing to the increasing cesarean delivery rate. *Obstet Gynecol*. 2011;118:29–38.
24. Draycott TJ, Crofts JF, Ash JP, et al. Improving neonatal outcome through practical shoulder dystocia training. *Obstet Gynecol*. 2008;112:14–20.
25. Boulvain M, Irion O, Dowswell T, et al. Induction of labour at or near term for suspected fetal macrosomia. *Cochrane Database Syst Rev*. 2016;5:CD000938.
26. Rozenberg P. In case of fetal macrosomia, the best strategy is the induction of labor at 38 weeks of gestation. *J Gynecol Obstet Biol Reprod (Paris)*. 2016;45:1037–1044.
27. Osterman MJ, Martin JA. *Recent Declines in Induction of Labor by Gestational Age*. Hyattsville, MD: National Center for Health Statistics; 2014.
28. Magro-Malosso ER, Saccone G, Chen M, et al. Induction of labour for suspected macrosomia at term in non-diabetic women: a systematic review and meta-analysis of randomized controlled trials. *BJOG*. 2017;124:414–421.
29. Chauhan SP, Blackwell SB, Ananth CV. Neonatal brachial plexus palsy: incidence, prevalence, and temporal trends. *Semin Perinatol*. 2014;38:210–218.

Copyright © 2017 Wolters Kluwer Health, Inc. All rights reserved.

EXHIBIT 5

SCIENTIFIC ARTICLE

Assessment of Current Epidemiology and Risk Factors Surrounding Brachial Plexus Birth Palsy

Joshua M. Abzug, MD,* Charles T. Mehlman, DO, MPH,† Jun Ying, PhD‡

Purpose Brachial plexus birth palsy (BPBP) is common; however, the current incidence is unknown and more than 50% of infants with BPBP have no known risk factors. The purpose of this study was to determine the current incidence of BPBP, assess known risk factors, and evaluate hypotonia as a new risk factor, as well as estimate the length of stay (LOS) and direct costs of children with an associated BPBP injury.

Methods Data from the 1997 to 2012 Kids' Inpatient Database data sets were evaluated to identify patients with a BPBP injury and various risk factors. Evaluation of LOS data and direct costs was also performed. Multivariable logistic regression analysis was utilized to assess the association of BPBP with its known and previously undescribed risk factors.

Results The incidence of BPBP has steadily decreased from 1997 to 2012, with an incidence of 0.9 ± 0.01 per 1,000 live births recorded in 2012. Shoulder dystocia is the number 1 risk factor for the development of a BPBP injury. Hypotonia is a newly recognized risk factor for the development of BPBP. Fifty-five percent of infants with BPBP have no known perinatal risk factors. The initial hospital LOS is approximately 20% longer for children with a BPBP injury and the hospital stay direct costs are approximately 40% higher.

Conclusions The incidence of BPBP is decreasing over time. Shoulder dystocia continues to be the most common risk factor for sustaining a BPBP injury. Children with a BPBP injury have longer LOSs and hospital direct costs compared with children without a BPBP injury. *(J Hand Surg Am. 2018;■(■):1.e1-e10. Copyright © 2018 by the American Society for Surgery of the Hand. All rights reserved.)*

Type of study/level of evidence Prognostic II.

Key words Brachial plexus birth palsy (BPBP), obstetrical brachial plexus injury (OBPI), risk factors, shoulder dystocia.

Online Exclusive

From the *Department of Orthopaedics, University of Maryland School of Medicine, Timonium, MD; the †Department of Orthopaedics, and the ‡Department of Internal Medicine, University of Cincinnati College of Medicine, Cincinnati, OH.

Received for publication November 6, 2017; accepted in revised form July 30, 2018.

This study was funded by MITRE corporation.

Corresponding author: Joshua M. Abzug, MD, Department of Orthopaedics, University of Maryland School of Medicine, 1 Texas Station Ct., Suite 300, Timonium, MD 21093; e-mail: jabzug@umoa.umm.edu.

0363-5023/18/■■-0001$36.00/0
https://doi.org/10.1016/j.jhsa.2018.07.020

Brachial plexus birth palsy (BPBP) is common, reportedly occurring from 0.38-5.1 per 1,000 live births in various countries.[1,2] The incidence in the United States has been shown to be 1.51 per 1,000 live births[3]; however, this is from data obtained between 1997 and 2003. Although several known risk factors exist, more than 50% of infants with BPBP have no known risk factors.[3] Furthermore, many of the known risk factors, including shoulder dystocia and the use of vacuum or forceps during delivery, may be less relevant as the rate of cesarean delivery has steadily increased over the past 2 decades.[4–6] In addition, cesarean delivery has been shown to be protective for BPBP injuries.[3,7] Alternative risk factors likely exist accounting for the large number of patients with no currently known risk factors and the fairly steady rate of BPBP injuries, despite the increase in cesarean delivery rates.[3,4,6] For example, relative hypotonia has been thought to make the brachial plexus more susceptible to stretch during delivery.[8,9] Therefore, it is important to understand the current incidence of BPBP as well as the current associated risk factors for infants born in the United States.

Once a BPBP injury has occurred, the initial treatment is observation and passive range of motion exercises because 80% to 95% of children will recover spontaneously within the first 2 months of life and, therefore, have no long-term sequelae.[10,11] The children who do not spontaneously recover by 2 months of age will likely have permanent loss; however, they do not require any immediate treatment regarding the BPBP injury. Despite this, children sometimes undergo additional monitoring in the hospital/neonatal intensive care unit or have various precautions placed on the limb. There are associated increases in direct costs when this happens, although the need for this is unclear, given that additional monitoring and/or precautions placed on the limb will not alter the extent of injury or recovery. The time an infant stays in the hospital following a birth when a BPBP injury occurs, and the direct cost associated with the stay, is currently unknown.

The purpose of this study was to identify the current incidence of BPBP injuries in the United States using the nationwide Kids' Inpatient Database. In addition, assessment of the previously known risk factors was performed, as well as a specific assessment of hypotonia. Lastly, an assessment of the length of stay (LOS) and direct costs associated with infants who have a BPBP injury was also performed.

PATIENT AND METHODS

Data source

The data for this study were obtained from a national public database, developed as part of the Healthcare Cost and Utilization Project, which is a federal-state-industry partnership sponsored by the Agency for Healthcare Research and Quality. This database is available for purchase and represents a sample of weighted pediatric discharges from all non-rehabilitation community hospitals in states participating in the Healthcare Cost and Utilization Project. These hospitals have changed over the course of the study; however, the database has maintained the ability to be a representative sample of pediatric inpatient care.[12] For the purposes of this database, community hospitals are defined as nonfederal, short-term (< 30-day-stay) hospitals that are available to the public and include both academic medical centers and specialty hospitals. The specialty hospitals include obstetrics-gynecology; ear, nose, and throat; orthopedic; and pediatric services. To ensure an accurate case mix in the database, the discharges are sorted by state, hospital, Diagnosis-Related Group, and a random number within each Diagnosis-Related Group. Subsequently, systematic random sampling selects 10% of uncomplicated in-hospital births and 80% of complicated in-hospital births and other pediatric cases from each participating hospital.[12] International Classification of Diseases, Ninth Revision (ICD-9), codes used for complicated and uncomplicated births have been consistent throughout the study period. Therefore, the Kids' Inpatient Database provides sampling weights for obtaining national estimates.

TABLE 1. Hospital Characteristics Available in the Kids' Inpatient Database

Hospital Characteristic	Variables
Location	Urban, rural
Region	West, northeast, midwest, south
Bed size	Small, medium, large
Teaching status	Teaching, no teaching

The Kids' Inpatient Database is released every 3 years, most recently in 2012.[13] All existing databases, a total of 6 from 1997 to 2012, were used in this study. Two types of data were assessed for each database: inpatient stay core data and hospital data. The inpatient core data contain demographic information, clinical information, and hospital payment information, whereas the hospital data contain information on each hospital characteristic (Table 1). The 2 datasets were then merged by hospital identifier to create a working dataset for analyses. This study was reviewed by an institutional review board and was deemed exempt owing to the deidentified nature of the database.

TABLE 2. International Classification of Diseases, Ninth Revision Codes Used

Disease	ICD-9 Code
BPBP	767.6
Shoulder dystocia	763.1
Use of forceps during delivery	763.2
Use of vacuum during delivery	763.3
Breech delivery	763.0
Cesarean delivery	V30.01, V31.01, V33.01, V34.01, V37.01, V39.01, 763.4
Exceptionally large baby (an infant weighing > 4.5 kg)	766.0
Heavy for dates (a fetus or infant large for dates, regardless of period of gestation)	766.1
Twin or multiple birth mates	V31.00, V31.01, V32.00, V32.01, V33.00, V33.01, V34.00, V34.01, V35.00, V35.01, V36.00, V36.01, V37.00, V37.01
Hypotonia	358.8, 359.0, 728.9, 781.3, 779.89

TABLE 3. Summary of Demographic and Health-Care Information of Live-Born Infants

Variable	1997	2000	2003	2006	2009	2012	All
Total n of infants*	3,915,210	4,004,783	4,012,392	4,205,436	4,103,115	3,918,489	24,159,426
Sex†							
Female	48.60 ± 0.10	48.84 ± 0.10	48.57 ± 0.08	48.66 ± 0.08	48.66 ± 0.07	48.67 ± 0.07	48.66 ± 0.03
Race†							
White	60.94 ± 0.93	57.99 ± 1.08	52.99 ± 0.93	51.79 ± 0.84	52.10 ± 0.75	52.43 ± 0.70	54.56 ± 0.55
Black	15.13 ± 0.69	12.97 ± 0.69	12.56 ± 0.44	12.90 ± 0.42	13.78 ± 0.41	14.22 ± 0.39	13.60 ± 0.30
Hispanic	16.22 ± 0.70	19.70 ± 0.87	23.67 ± 0.96	24.61 ± 0.90	22.82 ± 0.78	20.10 ± 0.63	21.22 ± 0.54
Asian or PI	3.45 ± 0.26	3.76 ± 0.28	4.25 ± 0.31	4.44 ± 0.26	4.65 ± 0.22	5.51 ± 0.29	4.39 ± 0.17
Native American	0.51 ± 0.12	0.48 ± 0.06	0.48 ± 0.05	0.61 ± 0.05	0.91 ± 0.08	0.91 ± 0.09	0.66 ± 0.04
Other	3.75 ± 0.30	5.11 ± 0.41	6.05 ± 0.43	5.66 ± 0.32	5.73 ± 0.28	6.84 ± 0.40	5.58 ± 0.18
Primary expected payer†							
Medicare	0.12 ± 0.03	0.18 ± 0.04	0.13 ± 0.03	0.14 ± 0.04	0.13 ± 0.03	0.35 ± 0.06	0.17 ± 0.02
Medicaid	34.36 ± 0.68	34.47 ± 0.74	38.94 ± 0.72	42.49 ± 0.67	45.83 ± 0.64	46.78 ± 0.60	40.54 ± 0.42
Private (HMO)	56.22 ± 0.80	57.82 ± 0.89	53.16 ± 0.77	49.58 ± 0.74	47.07 ± 0.68	45.76 ± 0.62	51.57 ± 0.48
Self-pay	5.28 ± 0.26	4.60 ± 0.25	4.79 ± 0.26	5.28 ± 0.26	4.27 ± 0.19	3.86 ± 0.17	4.68 ± 0.13
No charge	0.26 ± 0.08	0.38 ± 0.14	0.14 ± 0.05	0.18 ± 0.04	0.16 ± 0.04	0.08 ± 0.02	0.20 ± 0.04
Other	3.65 ± 0.38	2.55 ± 0.39	2.84 ± 0.27	2.32 ± 0.13	2.53 ± 0.12	3.17 ± 0.14	2.83 ± 0.12

HMO, Health Maintenance Organization; PI, Pacific Islander.
*The values are weighted estimates of total number of infants from the Kids' Inpatient Database.
†The values are weighted estimates of the mean frequency and the standard error (in percentage) from the Kids' Inpatient Database.

Study variables

The primary outcome of interest was the dichotomous variable of BPBP being present or absent. This was identified in the database utilizing the ICD-9 code 767.6. The known risk factors associated with a BPBP, as well as potentially new risk factors, such as hypotonia, were also identified using ICD-9 codes (Table 2). Characteristics associated with the hospital (Table 1) were also assessed. The LOS data and direct costs in U.S. dollars were recorded. Analysis was performed for each individual dataset time point (1997, 2000, 2003, 2006, 2009, and 2012) as well as

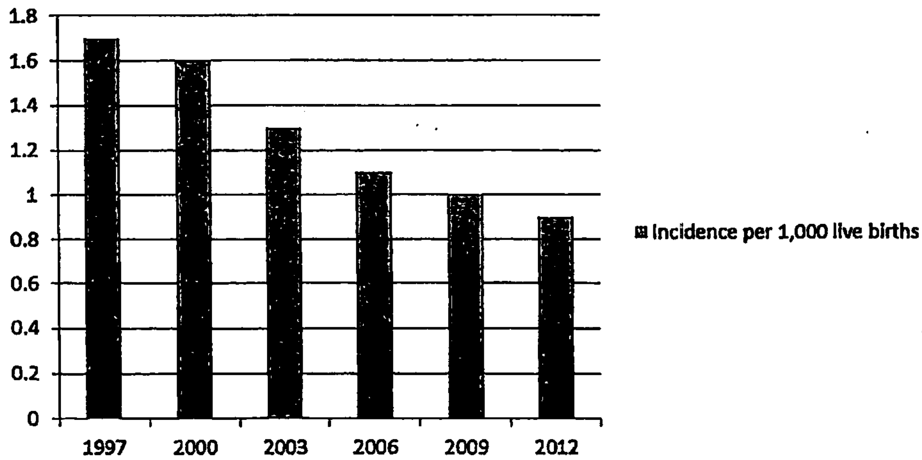


FIGURE 1: Incidence of BPBP injury per 1,000 live births over the time period of 1997 to 2012.

TABLE 4. Summary of the Incidence of BPBP and its Risk Factors*

Variable	1997	2000	2003	2006	2009	2012	All
BPBP	0.17 ± 0.01	0.16 ± 0.01	0.13 ± 0.01†‡	0.11 ± 0.01†‡	0.10 ± 0.01†‡	0.09 ± 0.01†	0.13 ± 0.01
Forceps delivery	0.20 ± 0.02	0.10 ± 0.01†‡	0.08 ± 0.01†	0.04 ± 0.01†‡	0.05 ± 0.01†	0.005 ± 0.01†	0.09 ± 0.01
Use of vacuum	0.32 ± 0.03	0.26 ± 0.04	0.20 ± 0.01†	0.18 ± 0.01†	0.24 ± 0.01†‡	0.28 ± 0.02	0.25 ± 0.01
Cesarean delivery	19.89 ± 0.16	22.49 ± 0.17†‡	27.11 ± 0.18†‡	30.92 ± 0.19†‡	32.53 ± 0.20†‡	32.32 ± 0.18†	27.61 ± 0.13
Breech delivery	0.13 ± 0.01	0.12 ± 0.01	0.09 ± 0.01†	0.11 ± 0.01	0.13 ± 0.01	0.16 ± 0.01	0.12 ± 0.01
Shoulder dystocia	0.26 ± 0.02	0.23 ± 0.02	0.20 ± 0.01†	0.20 ± 0.01†	0.23 ± 0.01‡	0.26 ± 0.01	0.23 ± 0.01
Exceptionally large baby	0.55 ± 0.02	0.50 ± 0.02	0.42 ± 0.01†‡	0.31 ± 0.01†‡	0.27 ± 0.01†‡	0.24 ± 0.01†‡	0.38 ± 0.01
Heavy for dates	4.79 ± 0.15	4.86 ± 0.17	5.07 ± 0.13	4.96 ± 0.12	5.14 ± 0.12	5.33 ± 0.12	5.02 ± 0.08
Twin or multiple birth	2.53 ± 0.05	2.83 ± 0.05†‡	3.07 ± 0.05†	3.12 ± 0.05†	3.19 ± 0.05†	3.14 ± 0.04†‡	2.98 ± 0.03
Hypotonia	0.03 ± 0.01	0.03 ± 0.01	0.51 ± 0.02†‡	3.28 ± 0.08†‡	4.22 ± 0.10†‡	4.40 ± 0.10†‡	2.10 ± 0.04

*The values are weighted estimates of the mean frequency and the standard error from the Kids' Inpatient Database.
†The frequencies (incidences) are significantly different from those in 1997 ($P < .05$).
‡The frequencies (incidences) are significantly different ($P < .05$) than the database from 3 years prior.

in a linear manner to assess for any possible trends over time. Adjusting factors for sociodemographic characteristics, such as sex, race, and payer status, were used for the analyses. Table 3 contains the breakdown of the various categories pertaining to race and payer status.

Statistical analysis

The BPBP and the potential risk factors were binary and/or categorical variables and, therefore, are summarized as frequencies or percentages. Multivariable logistic regression models were performed to assess associations of the binary dependent variable of BPBP with risk factors as the key independent variables of interest, after adjusting for other independent variables or covariates, such as the sociodemographic characteristics, hospital-based characteristics, and time. In addition, similar logistic regression and/or multinomial logistic regression models were performed to assess trends over time.

TABLE 5. Summary of Risk Factors Associated With BPBP

Risk Factor	Category	Number of Live-Born Infants With Injury	Number of Live-Born Infants	Frequency*	OR (95% CI)†
Shoulder dystocia	Yes	5,773	55,562	10.390 ± 0.324	116.01 (107.15–125.61)
	No	24,903	24,103,864	0.103 ± 0.002	1.00
Exceptionally large baby	Yes	1,638	91,784	1.785 ± 0.072	14.86 (13.59–16.26)
	No	29,038	24,067,641	0.121 ± 0.002	1.00
Forceps delivery	Yes	274	20,758	1.318 ± 0.120	10.13 (8.14–12.60)
	No	30,402	24,138,667	0.126 ± 0.002	1.00
Vacuum delivery	Yes	579	59,473	0.974 ± 0.059	7.65 (6.67–8.76)
	No	30,096	24,099,953	0.125 ± 0.002	1.00
Heavy for dates	Yes	7,744	1,213,825	0.638 ± 0.013	7.05 (6.73–7.39)
	No	22,932	22,945,601	0.100 ± 0.001	1.00
Hypotonia	Yes	950	506,460	0.188 ± 0.008	1.93 (1.76–2.12)
	No	506,460	23,652,966	0.126 ± 0.002	1.00
Breech delivery	Yes	52	29,909	0.175 ± 0.032	1.34 (0.90–1.99)
	No	30,624	24,129,516	0.127 ± 0.002	1.00
Twin or multiple birth mates	Yes	203	720,837	0.028 ± 0.003	0.21 (0.17–0.27)
	No	30,472	23,438,589	0.130 ± 0.002	1.00
Cesarean delivery	Yes	1,266	6,670,357	0.019 ± 0.001	0.12 (0.11–0.13)
	No	29,410	17,489,068	0.168 ± 0.002	1.00

95% CI, 95% confidence interval.

*The values given are a mean percentage and the standard error of the mean. All estimates are from a logistic regression model adjusting for sociodemographic characteristics, hospital-based characteristics, and the time effect.

†A larger (smaller) than 1 lower bound (upper bound) of the 95% confidence interval indicates a significantly larger (smaller) than 1 OR ($P < .05$), with use of a logistic regression model adjusting for sociodemographic characteristics, hospital-based characteristics, and the time effect.

The estimates of frequencies (or incidences) obtained from the logistic regression models were adjusted by a sampling weight, based on the discharge information, to account for the probability of selection of each observation and ensure unbiased estimates for the representative population. Variances of frequencies (or incidences) were estimated with the use of a Taylor series linearization (generalized estimation equation) method to account for within-cluster correlation in the multistage complex sampling design of the Kids' Inpatient Database. When assessing LOS and direct cost, log transfers were performed to correct skewness of the data. A *P* value of .05 or less was considered significant.

RESULTS

A combined total of 24,159,426 live births was identified from the years 1997, 2000, 2003, 2006, 2009, and 2012. A slight majority of children were white (55%), males (51%), with private insurance (52%) (Table 3). The total number of documented BPBP patients was 31,407. The mean incidence (standard error) of BPBP injuries for the 15 years covered by the database was 1.3 (0.01) per 1,000 live births. The rate steadily decreased from 1.7 per 1,000 live births in 1997 to 1.3 per 1,000 live births in 2003 to 0.9 per 1,000 live births in 2012 (Fig. 1).

The incidences of BPBP and its risk factors are listed in Table 4. As the incidence of cesarean delivery increased, the incidence of BPBP injury declined (Table 4). The risk factors associated with BPBP injuries are assessed in Table 5. The largest risk factor was shoulder dystocia with an odds ratio (OR) of 116.0, thus indicating that a child with shoulder dystocia has a 10.4% probability of sustaining a BPBP injury compared with a 0.10%

TABLE 6. Risk Factors as a Predictor of BPBP

Risk Factor	Frequency of Infant With BPBP and a Risk Factor (n = 30,676)	Infants With BPBP and Risk Factor (%)
Breech delivery (a)	52	0.17
Shoulder dystocia (b)	5,773	18.82
Exceptionally large baby (> 4.5 kg) (c)	1,638	5.34
Heavy for dates (d)	7,744	25.24
Hypotonia (g)	950	3.10
(a)–(d)	12,940	42.18
Forceps (e)	274	0.89
Vacuum delivery (f)	579	1.89
(e)–(f)	838	2.73
(a)–(f)	13,411	43.72
(a)–(g)	13,901	45.32

The letters in parentheses (a-g) correspond to the 7 risk factors.

chance of a child sustaining a BPBP injury without shoulder dystocia. Hypotonia was also found to be a risk factor with an OR of 1.93. Cesarean delivery and twin or multiple birth mates were protective against BPBP injury, with ORs of 0.12 and 0.21, respectively (Table 5).

The percentage of infants with a BPBP injury and a risk factor are presented in Table 6. Only 43.7% of children had one or more previously known risk factors (breech delivery, shoulder dystocia, exceptionally large baby, heavy for dates, forceps delivery, or vacuum delivery). This increased to 45.3% with the inclusion of hypotonia as a risk factor (Table 6).

The LOS was found to be significantly longer ($P < .05$) in patients with a BPBP injury compared with those who did not have a BPBP injury. The average increase in LOS was approximately 20% longer for infants with a BPBP injury than for those who did not have a BPBP injury (Table 7). Correspondingly, the average direct cost of the hospital stay was approximately 40% higher in children with BPBP injuries than in those without BPBP injuries (Table 7). Both LOSs and the corresponding direct costs were higher in large hospitals and urban teaching hospitals, compared with small- and medium-sized hospitals and rural and urban nonteaching hospitals, respectively (Table 8). In addition, Medicare and Medicaid patients have the longest LOSs and highest direct costs compared with private insurance, self-pay, and no-charge patients (Table 8).

DISCUSSION

The present study assessed the current incidence and risk factors surrounding BPBP in the United States utilizing the Kids' Inpatient Database, which had data for 24,159,426 live births over a 15-year period. The largest previous study performed to date utilized the first 3 datasets from the Kids' Inpatient Database (1997, 2000, and 2003) and found a 1.5 per 1,000 live birth incidence of BPBP injury.[3] The authors of that study noted a decreasing trend in BPBP injuries. In this study, the incidence of BPBP injury was found to be decreasing over the 15-year period, with an incidence of 0.9 per 1,000 live births present in 2012. This likely corresponds to the increasing rate of cesarean delivery over the past 2 to 3 decades,[4–6] as was demonstrated by an increasing incidence from 19.9% in 1997 to an incidence of 32.3% in 2012 in the current study (Table 4). Furthermore, cesarean delivery was shown to be protective against a BPBP injury (Table 5). Additional causes for the decreasing incidence of BPBP may be the decreasing rate of exceptionally large babies (decreased from an incidence of 0.55% in 1997 to 0.24% in 2012), the decreasing use of forceps during delivery (decreased from 0.20% in 1997 to 0.05% in 2012), and the increasing rate of twin or multiple birth mates (increased from 2.53% in 1997 to 3.14% in 2012), which has a protective effect against a BPBP injury (Tables 4, 5).

Many known risk factors exist can lead to a BPBP injury. including shoulder dystocia, the use of forceps during delivery, the use of a vacuum during delivery, breech delivery, an exceptionally large baby (>4.5 kg), and a baby heavy for dates. However, in our study, only 43.7% of patients with a BPBP injury had 1 or more of these risk factors (Table 6). Hypotonia has been proposed as a potential risk factor owing to the possibility of making the plexus more susceptible to stretch during the delivery process.[4,7] The current study found that hypotonia is a risk factor for BPBP injury with an OR of 1.93 (Table 5). When hypotonia was added to the other known risk factors, 45.3% of patients had 1 or more identifiable risk factors; however, approximately 55% of cases were still not associated with a risk factor (Table 6).

TABLE 7. Total Charges and LOS

Year	BPBP Injury Total Charges (in U.S.$) Mean (95% CI) (A)	No BPBP Total Charges (in U.S.$) Mean (95% CI) (B)	Fold (A/B)	BPBP Injury LOS (in d) Mean (95% CI) (C)	No BPBP injury LOS (in d) Mean (95% CI) (D)	Fold (C/D)
1997	3,059 (2,862–3,269)	1,835 (1,783–1889)	1.67‡	2.3 (2.2–2.4)	1.8 (1.8–1.9)	1.25‡
2000	3,282 (3,075–3,502)	2,039 (1,977–2,103)*†	1.61‡	2.4 (2.3–2.5)	2.0 (2.0–2.0)*†	1.21‡
2003	3,983 (3,779–4,199)*†	2,577 (2,507–2,649)*†	1.55‡	2.4 (2.3–2.5)*†	2.1 (2.1–2.1)*†	1.15‡
2006	4,679 (4,421–4,952)	3,012 (2,928–3,099)*†	1.55‡	2.5 (2.4–2.6)*	2.2 (2.1–2.2)*†	1.16‡
2009	5,706 (5,368–6,066)*†	3,514 (3,421–3,610)*†	1.62‡	2.5 (2.4–2.6)*	2.2 (2.1–2.2)*	1.15‡
2012	7,140 (6,673–7,640)*†	4,125 (4,009–4,244)*†	1.73‡	2.6 (2.5–2.7)*	2.2 (2.2–2.2)*	1.19‡

95% CI, 95% confidence interval.
The letters in parentheses (A-D) correspond to the 4 categories.
*Indicates the difference against 1997 is significant with $P < .05$.
†Indicates the difference against the previous survey (3 y prior) is significant with $p < .05$.
‡Indicates the difference between BPBP injury and no injury is significant with $P < .05$.

Following a BPBP, the damage to the nerves represents a neuropraxia, axonotmesis, or neurotmesis injury.[11] To our knowledge, there are no known reports of further damage occurring to the nerves during the immediate postnatal period. Therefore, in our opinion, no additional precautions and/or monitoring are necessary related to a BPBP injury while the child is in the hospital and the usual discharge protocols can be followed, with the parents instructed to do the passive range of motion home exercises. However, the current study found an approximately 20% longer LOS and a corresponding 40% increase in direct cost for children with a BPBP injury compared with those who did not have a BPBP injury. Although we cannot know whether these findings are directly related to the BPBP injury, the association warrants further investigation. It is possible that better education of providers regarding the initial management of these injuries may lead to decreases in LOSs and the associated direct costs. Alternatively, it is possible that children with BPBP have other factors leading to an increased LOS and increase in direct cost that we did not identify.

Certain hospital characteristics were found to be associated with longer LOSs and increased direct costs. These included larger hospitals, compared with small- and medium-sized hospitals, and urban teaching hospitals, compared with rural and urban nonteaching hospitals (Table 8). The factors surrounding these increased LOSs and direct costs are not clear; however, we feel this may be due to the larger hospitals and urban teaching hospitals having more difficult obstetrical cases referred in. Further investigation is necessary to determine if LOS and direct cost can be decreased in these environments.

The present study has limitations that must be acknowledged. First and foremost, the data analyzed are from a large national database, which contains self-reported data from many institutions. Furthermore, the data extracted ICD-9 codes, which may have been entered incorrectly or had alternative codes utilized. The primary ICD-9 code utilized to perform the current study was 767.6, injury to the brachial plexus in the neonatal period. However, 2 other codes for brachial plexus injury exist, but these codes are typically reserved for older children. In addition, the actual BPBP injury may not have been confirmed by a neurologist, neurosurgeon, or hand/upper extremity surgeon and may have relied solely on the primary care provider. Lastly, hypotonia may have been coded during the discharge as a result of decreased tone in the affected extremity, as opposed to hypotonia being present in all 4 extremities.

Over the 15-year period studied, there was a decreasing incidence of BPBP. The presumed risk factor of hypotonia was confirmed to be a risk factor for children sustaining a BPBP injury. Lastly, the present study found that children with a BPBP injury have a longer LOS and higher associated direct costs with their initial hospital course. Future studies are needed to monitor the incidence of BPBP over time, look for additional risk factors, and determine ways to

TABLE 8. Comparison of Total Charges and LOS With Risk Factors for BPBP

Risk Factor	Category	Total Charge (in US$) With 95% CI	Fold	*P* Value	LOS (in d) With 95% CI	Fold	*P* Value
Forceps	No	3,981 (3,702–4,281)	1.00		2.31 (2.21–2.41)	1.00	
	Yes	4,675 (3,790–5,766)	1.17	.114	2.90 (2.47–3.40)	1.25	< .05
Vacuum delivery	No	3,970 (3,691–4,269)	1.00		2.31 (2.21–2.41)	1.00	
	Yes	4,735 (4,217–5,317)	1.19	< .05	2.54 (2.27–2.85)	1.10	.08
Cesarean delivery	No	3,878 (3,622–4,151)	1.00		2.26 (2.18–2.35)	1.00	
	Yes	6,984 (6,335–7,700)	1.80	< .05	3.69 (3.45–3.95)	1.63	< .05
Breech delivery	No	3,977 (3,699–4,277)	1.00		2.31 (2.21–2.41)	1.00	
	Yes	12,234 (8,889–16,387)	3.08	< .05	3.39 (1.99–5.79)	1.47	.16
Shoulder dystocia	No	3,766 (3,504–4,049)	1.00		2.29 (2.19–2.39)	1.00	
	Yes	4,926 (4,523–5,365)	1.31	< .05	2.41 (2.27–2.56)	1.05	< .05
Exceptionally large baby	No	3,943 (3,666–4,241)	1.00		2.30 (2.20–2.40)	1.00	
	Yes	4,722 (4,245–5,253)	1.20	< .05	2.64 (2.45–2.84)	1.15	< .05
Heavy for dates	No	3,906 (3,629–4,203)	1.00		2.30 (2.20–2.40)	1.00	
	Yes	4,188 (3,889–4,510)	1.07	< .05	2.35 (2.25–2.46)	1.02	< .05
Twin or multiple birth	No	3,965 (3,703–4,245)	1.00		2.30 (2.22–2.39)	1.00	
	Yes	7,163 (5,621–9,129)	1.81	< .05	4.27 (3.54–5.16)	1.86	< .05
Hypotonia	No	3,752 (3,462–4,066)	1.00		2.26 (2.16–2.37)	1.00	
	Yes	10,953 (9,960–12,046)	2.92	< .05	3.50 (3.21–3.80)	1.55	< .05
Female	No	4,235 (4,007–4,668)	1.00		2.39 (2.28–2.51)	1.00	
	Yes	3,667 (3,415–3,938)	0.85	< .05	2.23 (2.14–2.33)	0.93	< .05
Race	White	3,441 (3,237–3,657)	1.00		2.17 (2.08–2.25)	1.00	
	Black	3,747 (3,424–4,101)	1.09	.063	2.19 (2.05–2.32)	1.01	.99
	Hispanic	3,795 (3,549–4,059)	1.10	< .05	2.13 (2.05–2.23)	0.98	.99
	Asian	3,940 (3,592–4,322)	1.15	< .05	2.34 (2.23–2.46)	1.08	< .05
	Native American	5,178 (4,366–6,141)	1.50	< .05	2.97 (2.70–3.26)	1.37	< .05
	Other	3,997 (3,702–4,316)	1.16	< .05	2.18 (2.06–2.31)	1.01	.99

(Continued)

TABLE 8. Comparison of Total Charges and LOS With Risk Factors for BPBP (Continued)

Risk Factor	Category	Total Charge (in US$) With 95% CI	Fold	*P* Value	LOS (in d) With 95% CI	Fold	*P* Value
Primary expected payer	Medicare	4,806 (3,510–6,579)	1.00		2.51 (2.05–3.08)	1.00	
	Medicaid	4,255 (4,109–4,405)	0.89	.99	2.36 (2.31–2.42)	0.94	.99
	Private (HMO)	3,638 (3,512–3,768)	0.76	.99	2.19 (2.13–2.25)	0.87	.99
	Self-pay	3,356 (3,122–3,609)	0.70	.406	1.95 (1.84–2.07)	0.78	.270
	No charge	3,905 (3,706–4,115)	0.81	.99	2.41 (2.34–2.49)	0.96	.99
	Other	4,094 (3,306–5,070)	0.85	.99	2.50 (2.29–2.72)	0.99	.99
Hospital bed size	Small	3,291 (3,049–3,554)	1.00		2.06 (1.97–2.17)	1.00	
	Medium	4,033 (3,736–4,353)	1.23	< .001	2.34 (2.23–2.44)	1.13	< .001
	Large	4,759 (4,395–5,154)	1.45	< .001	2.57 (2.45–2.69)	1.24	< .001
Hospital location/teaching	Rural	2,648 (2,460–2,850)	1.00		1.86	1.00	
	Urban no Teaching	4,122 (3,838–4,439)	1.56	< .001	2.29 (2.19–2.39)	1.23	< .001
	Urban teaching	5,787 (5,329–6,284)	2.19	< .001	2.90 (2.77–3.05)	1.56	< .001
Hospital region	West	5,295 (4,938–5,678)	1.00		2.17 (2.08–2.26)	1.00	
	Northeast	4,116 (3,777–4,486)	1.29	< .001	2.36 (2.24–2.49)	0.92	< .001
	Midwest	3,762 (3,368–4,202)	1.41	< .001	2.33 (2.17–2.50)	0.93	.069
	South	3,069 (2,867–3,285)	1.73	< .001	2.39 (2.30–2.49)	0.91	< .001

95% CI, 95% confidence interval; HMO, Health Maintenance Organization.

decrease the LOSs and direct costs associated with newborns who sustain a BPBP injury.

REFERENCES

1. Hoeksma A, ter Steeg A, Nelissen R, van Ouwerkerk W, Lankhorst G, de Jong BA. Neurological recovery in obstetric brachial plexus injuries: an historical cohort study. *Dev Med Child Neurol.* 2004;46(2):76–83.
2. Adler JB, Patterson RL Jr. Erb's palsy. Long-term results of treatment in eighty-eight cases. *J Bone Joint Surg Am.* 1967;49(6):1052–1064.
3. Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the Unites States. *J Bone Joint Surg Am.* 2008;90(6):1258–1264.
4. Barber EL, Lundsberg LS, Belanger K, Pettker CM, Funai EF, Illuzzi JL. Indications contributing to the increasing cesarean delivery rate. *Obstet Gynecol.* 2011;118(1):29–38.
5. Sebastião YV, Womack L, Vamos CA, et al. Hospital variation in cesarean rates: contribution of individual and hospital factors in Florida. *Am J Obstet Gynecol.* 2015;214(1):123.e1–123.e18.
6. Walsh JM, Kandamany N, Shuibhne NN, Power H, Murphy JF, O'Herlihy C. Neonatal brachial plexus injury: comparison of incidence and antecedents between 2 decades. *Am J Obstet Gynecol.* 2011;204(4):324.e1–324.e6.
7. Coroneos CJ, Voineskos SH, Coroneos MK, et al. Obstetrical brachial plexus injury: burden in a publicly funded, universal healthcare system. *J Neurosurg Pediatr.* 2016;17(2):222–229.
8. Waters PM. Obstetric brachial plexus injuries: evaluation and management. *J Am Acad Orthop Surg.* 1997;5(4):205–214.
9. Abzug JM, Kozin SH. Evaluation and management of brachial plexus birth palsy. *Orthop Clin North Am.* 2014;45(2):225–232.
10. Greenwald AG, Schute PC, Shiveley JL. Brachial plexus birth palsy: a 10-year report on the incidence and prognosis. *J Pediatr Orthop.* 1984;4(6):689–692.
11. Sjöberg K, Erichs K, Bjerre I. Cause and effect of obstetric (neonatal) brachial plexus palsy. *Acta Paediatr Scand.* 1988;77(3):357–364.
12. Healthcare Cost and Utilization Project (HCUP). *Introduction to the HCUP KID's Inpatient Database (KID), 2003.* Rockville, MD: Agency for Healthcare Research and Quality; November 2015. Available at: www.hcup-us.ahrq.gov/db/nation/kid/kid_2003_introduction_rpt.jsp. Accessed September 18, 2018.
13. Healthcare Cost and Utilization Project (HCUP). *HCUP Kids' Inpatient Database (KID) for 1997, 2000, 2003, 2006, 2009, and 2012* [dataset]. Rockville, MD: Agency for Healthcare Research and Quality. Available at: www.hcup-us.ahrq.gov/kidoverview.jsp. Accessed September 18, 2018.

EXHIBIT 6

DOI: 10.5137/1019-5149.JTN.21339-17.2
Received: 27.07.2017 / Accepted: 18.10.2017
Published Online: 13.11.2017

Turk Neurosurg 28(5):783-791,2018

Original Investigation

Anatomical Variations of Brachial Plexus in Fetal Cadavers

Alparslan KIRIK[1], Senem Ertugrul MUT[2], Mehmet Kadri DANEYEMEZ[3], Halil Ibrahim SECER[4]

[1]Etimesgut Sehit Sait Erturk State Hospital, Neurosurgery Clinic, Ankara, Turkey
[2]University of Kyrenia, Faculty of Medicine, Visiting Professor, Near East University, Faculty of Medicine, Department of Neurology, Girne, Turkish Republic of Northern Cyprus
[3]University of Health Sciences, Gulhane Training and Research Hospital, Department of Neurosurgery, Ankara, Turkey
[4]University of Kyrenia, Faculty of Medicine, Visiting Professor, Near East University, Faculty of Medicine, Department of Neurosurgery, Girne, Turkish Republic of Northern Cyprus

This study has been presented as an oral presentation at the 25th Scientific Congress of the Turkish Neurosurgical Society between 22 and 26 April 2011 at Antalya, Turkey.

ABSTRACT

AIM: To demonstrate the anatomical variations of the infantile brachial plexus.

MATERIAL and METHODS: A total of 20 plexus brachialis from 11 fetal cadavers were dissected and examined microscopically. The branching patterns and variations were evaluated. The width of the nerves was assessed at the level of the nerve root, trunk and cord on the basis of all brachial plexuses and they were arranged in terms of thickness.

RESULTS: Half of the brachial plexuses were found to be prefixed, while 15% were found to be postfixed. Truncus superior, medial cord and nervus ulnaris were found in normal formation, whereas anatomical variations were detected in the rest of the structures. The plexus brachialis elements were arranged in the following order from large to small according to their average thicknesses: C7>C6>C8>C5=T1; TS>TI>TM; PC>LC>MC.

CONCLUSION: Since the risk of injury for variated branches is higher, understanding the anatomical variations of plexus brachialis and its extensions are of significant importance during surgical intervention.

KEYWORDS: Anatomical variation, Brachial plexus, Fetus

ABBREVIATIONS: BP: Brachial plexus, **C:** Cervical root, **T:** Thoracic root, **TS:** Truncus superior, **TSdp:** Division posterior of truncus superior; **TM:** Truncus medius, **TMda:** Division anterior of truncus medius, **TMdp:** Division posterior of truncus medius, **TI:** Truncus inferior, **TIdp:** Division posterior of truncus inferior, **LC:** Lateral cord, **MC:** Medial cord, **PC:** Posterior cord, **Cl:** Clavicula, **SC:** Musculus subclavius, **SCM:** Musculus sternocleidomastoideus, **SA:** Musculus serratus anterior, **PMj:** Musculus pectoralis major, **PMn:** Musculus pectoralis minor, **GT:** Glandula troidea, **Tr:** Trachea, **AA:** Arteria axillaris, **VA:** Vena axillaris, **A:** Nervus axillaris, **M:** Nervus medianus, **U:** Nervus ulnaris, **MCT:** Nervus musculocutaneus, **R:** Nervus radialis, **CBM:** Nervus cutaneus brachi medialis, **DS:** Nervus dorsalis scapula, **SS:** Nervus suprascapularis, **SSS:** Nervus subscapularis superior, **SSI:** Nervus subscapularis inferior, **TD:** Nervus thoracodorsalis, **CABM:** Nervus cutaneus antebrachi medialis, **PL:** Nervus pectoralis lateralis, **PM:** Nervus pectoralis medialis, **TL:** Nervus thoracicus longus, **P:** Phrenic nerve.

Corresponding author: Halil Ibrahim SECER
E-mail: hisecer@yahoo.com

INTRODUCTION

The brachial plexus (BP) is an important structure due to its anatomical location and its vulnerability to damage. There are several variations in the formation and branching pattern. Therefore, knowledge of the anatomy and variations of BP is important in reducing the neurological damage that can be caused by surgical intervention (1,6).

BP is mostly formed by the anterior rami of C5-C8 and T1 spinal nerves and bifurcates into the upper (C5, C6 spinal nerves), middle and lower trunks (C7 and the union of C8, T1 spinal nerves). After the formation of trunks, the anterior and posterior divisions continue and they lead to the lateral cord, medial cord and posterior cord. After integration of the two anterior divisions of the upper truncus and middle truncus, the lateral cord is formed. The anterior division of the lower truncus continues as the medial cord. The fusion of the posterior divisions of the three trunks forms the posterior cord. Later, the cords give off terminal nerve branches that supply the upper extremities (7).

It has been demonstrated in studies that extensive variations may accompany BP in the formation of roots, trunks, divisions and cords. For instance, a C4 nerve supernumerary root was found or the T2 nerve root formed the BP (27). Studies conducted by Singhal et al., Matejcik, and Fazan et al., reported an incidence of prefixed BP at a proportion of 24%, 21.7% and 48%, respectively (4,14,23). On the other hand, postfixed BPs were reported by Singhal et al. and Matejcik et al. with a frequency of 5% and 2%, respectively (14,23). In general, variations are not limited to the origin of the BP, although division, cord and distal ramification anomalies have been also reported (27).

There are few research studies that have investigated fetal brachial plexuses. Studies by Uysal et al. and Uzmansel et al. have conducted morphological examinations of BP in the fetal period (24,25). Due to the limited number of investigations, the aim of this study was to present BP variations in fetal cadavers.

MATERIAL and METHODS

The study was performed at the microsurgery laboratory of the Neurosurgery Department of Gulhane Military Medical Academy with the approval of the Ankara 7th Ethical Committee (dated 13.01.2010, with decision number 22). All of the material used in the study belonged to the pathology department. A Zeiss Universal S3 operation microscope and a Sony W-290 digital camera were used. This anatomical study was conducted to assess the anatomical variations of the brachial plexus formation in 20 BPs from 11 fetuses sent for pathologic investigation from the gynecology and obstetrics clinic to the pathology laboratory at Gulhane Military Medical Academy Hospital. All the materials were aborted due to intrauterine death. Bilateral brachial plexus dissection was performed on 9 of the fetal cadavers. In 2 fetal cadavers, the anatomical structure of the left brachial plexus had been damaged before the study, which meant that only the right brachial plexuses were dissected. Fetuses were 23-32 (mean 28) weeks old and weighed between 460 and 1,470 (mean 923,64) grams (Table I).

The operating microscope was used in all dissections. The same incision that was described by MacCarty, Dunkerton, Hentz, Laurent and Ochiani was used before visualization of the brachial plexus and the branches in the plexus brachialis surgery (3,8,10,12,18). The skin incision was performed in three steps for dissection. The initial incision was made starting from the mastoid overhang through the lateral side of the sternocleidomastoid muscle, ending at the medial aspect of the clavicle. The second skin incision began at the end of the first incision, through the superior clavicle line, at which point the incision was oriented below to fit to the deltopectoral sulcus and was extended to the midpoint of the inner side of the arm. The third incision was made from the deltopectoral sulcus to the axillary fossa (Figure 1). The skin flap, fascia cervicalis superficialis, fascia cervicalis profunda and platysma muscle were all removed. The sternocleidomastoid muscle was cut at the insertion point of the clavicula and sternum. After removal of the sternocleidomastoid and omohyoid muscles, the clavicula was taken out by cutting the nearby articulatio acromioclavicularis and articulatio sternoclavicularis. Additionally, the pectoralis major and pectoralis minor muscles were removed by cutting close to their insertion points in order to visualize the divisions of the BP in the infraclavicular fossa. The anterior and median scalene muscles and all the tissues positioned in front of the C4-T2 vertebra levels were excised for the purpose of visualizing the nerve roots that composed the BP. The axillary subclavian artery and vein as well as all their branches were removed. During the dissection, the right and left BP, their branches, as well as their variations, were evaluated.

Furthermore, the thickness of the ramus ventralis nervi spinalis, trunks and cords were measured in millimeters. The thickness of the ramus ventralis nervi spinalis was measured

Table I: Gender, Gestational Age and Weight of Fetuses

Fetus No	Gender	Gestational age (weeks)	Weight (gram)
1	M	28	910
2	M	26	820
3	F	27	850
4	M	30	1220
5	F	30	1180
6	F	30	1140
7	F	32	1470
8	M	24	550
9	M	23	460
10	F	26	760
11	M	28	800

M: Male, F: Female.

from the intervertebral foramen exit point. The thicknesses of the other neural structures were measured at the level of their formation.

■ RESULTS

In the present study, a total of 20 plexuses from 6 male and 5 female fetal cadavers were examined. All BPs were located between the scalene anterior and scalene medius muscles and were united to form a truncus. BP was found to be prefixed in 10 BPs (Figure 1A), while there was no C4 involvement in the others. Among the BPs, 15% were found as postfixed plexus. In all of these postfixed BP cases, prefixed BP was also observed (Figure 1A).

Trunk Anomalies

In 10 BPs, C4, C5 and C6 roots formed the truncus superior (TS), while in the remaining 10 BPs, the C5 and C6 roots formed the TS. The truncus medius (TM) consisted of the C7 root and no variations were found in any of the BPs. In 17 cases (85%), the C8 and T1 roots formed the truncus inferior (TI), while in 3 (15%) cases, the C8, T1 and T2 roots formed the TI.

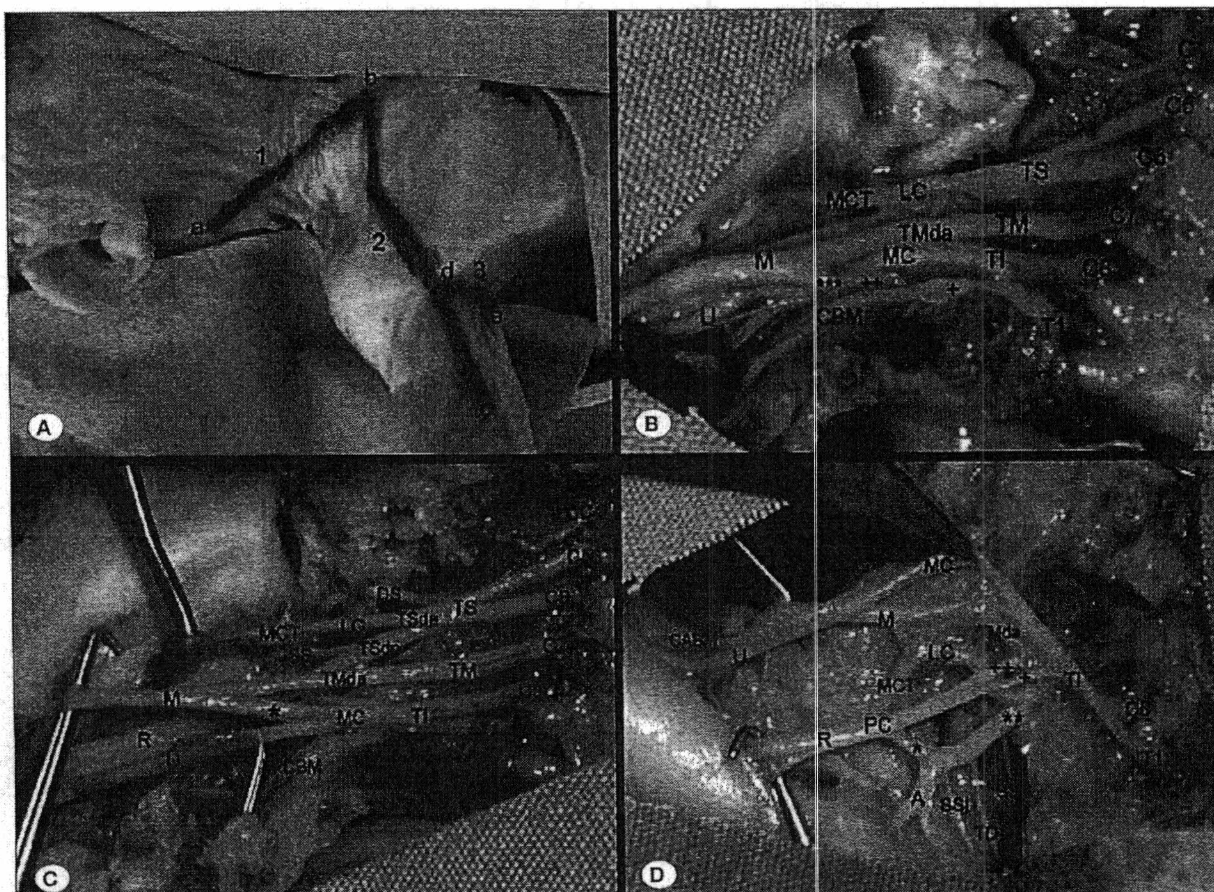


Figure 1: A) This figure shows three incisions of skin for dissection. The initial incision is between points a and b through the first line. The second incision is between points b and c through the second line. The last incision is between points d and e through the third line. **B)** Right BP: a branch from the C4 spinal nerve (*) joins the C5 spinal nerve (prefixed plexus). A branch of the T2 spinal nerve (**) joins the T1 spinal nerve (postfixed plexus). The nervus medianus consists of the anterior division of the truncus medius and radix medialis nervi mediani (***). A branch of the T1 spinal nerve (+) separates into two parts; one part is the nervus cutaneus brachi medialis and the other part is the nervus cutaneus antebrachi medialis (++). **C)** The lateral cord originates only from the anterior division of the truncus superior. The nervus medianus consists of the anterior division of the truncus medius and radix medianus nervi median, which is a branch of the medial cord (*). The nervus suprascapularis originates from the posterior division of the truncus medius. **D)** Right BP: the medial cord is removed and lifted with a tool. The nervus subscapularis superior, nervus subscapularis inferior and nervus thoracodorsalis originate from the posterior division of the truncus superior (**). The posterior cord arises from posterior division of the truncus medius (++) and the truncus inferior (+), and an additional branch of the posterior division of the truncus superior (*). Nervus axillaris separates two parts.

Cord Anomalies

In 13 BPs (65%) lateral cords (LC) were found in the normal anatomical structure. In 7 BPs (35%), LCs were formed only by the TS (Figures 1, 2, 3, 4). In one of these 7 BPs (5%), the anterior division of the TM gave off a small branch to the LC. Medial cords (MC) in all BPs and posterior cords (PC) in 18 BPs (90%) were found in the normal anatomical structure. In 2 BPs (10%), variations were observed. In one of these, the posterior divisions of the TM and TI first formed the PC, and the posterior division of the TS joined with the PC after giving the nervus subscapularis superior and inferior (Figure 2). In the other BP, the posterior division of the TS first gave off the nervus thoracodorsalis, then formed the PC with the posterior divisions of the TM and TI (Figure 1).

Distal Ramification Anomalies

In all BPs, the nervus musculocutaneous originated from the LCs. In only one BP (5%), the musculocutaneous nerve received a communicating branch from the anterior division of the TM (Figure 3).

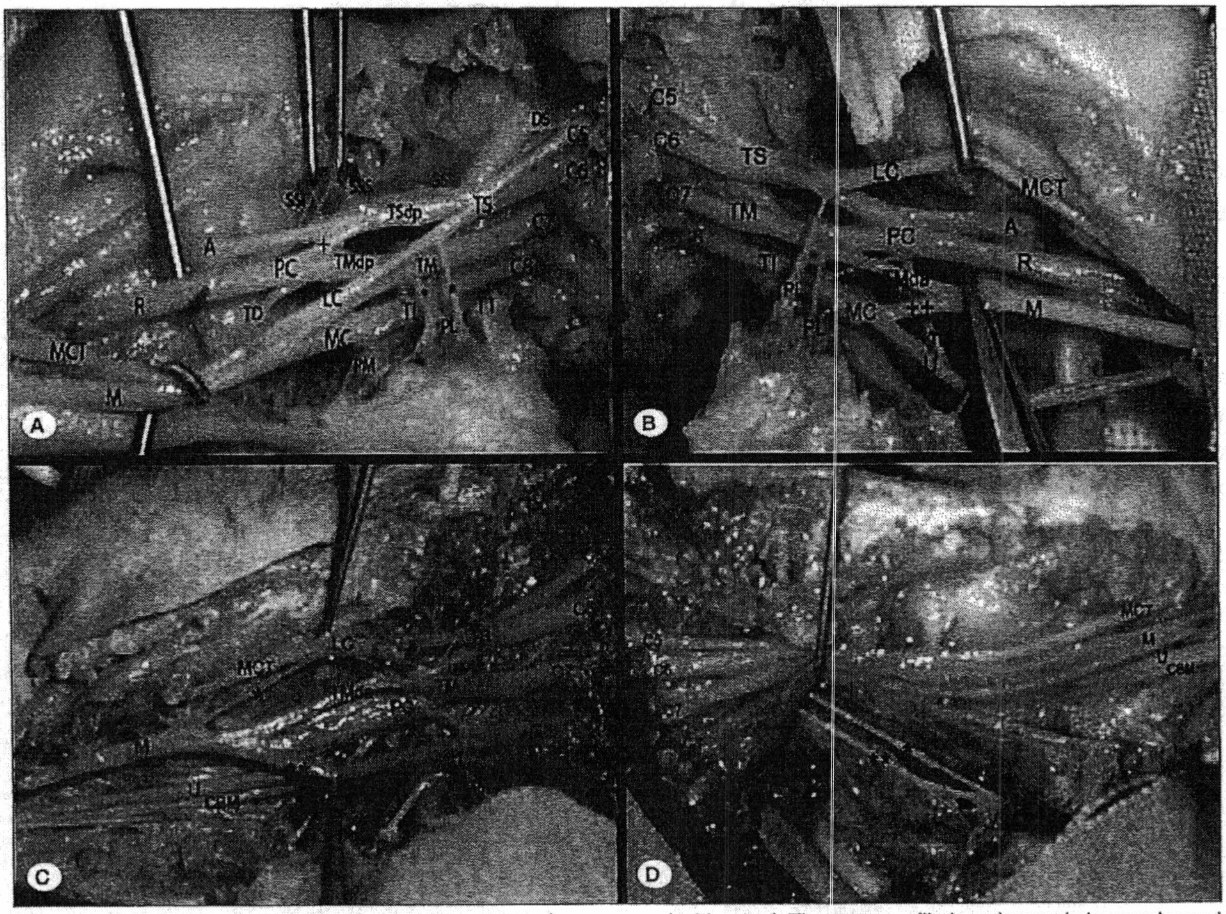


Figure 2: A) Right BP: the lateral cord and the medial cord are removed with a tool. The nervus axillaris, subscapularis superior and subscapularis inferior originate only from the posterior division of the truncus medialis. The nervus radialis and thoracodorsalis originate from the posterior cord. An additional branch of the posterior division of the truncus superior joins the posterior cord (+). The nervus pectoralis lateralis consists of two roots which arise from the anterior division of the truncus superior and truncus medius (*). **B)** Left BP: there is a thin additional connection from the truncus inferior to the nervus radialis (*). The lateral cord originates only from the anterior division of the truncus superior. There are two pectoralis lateralis nerves, one of which arises from the anterior division of the truncus superior, and the other arises from the anterior division of the truncus medius. These nerves form a conjunction together. The nervus medianus consists of the radix medialis nervi mediani (++) and the anterior division of the truncus medius. (+): nervus pectoralis medialis. **C)** Right BP: a thin branch of the anterior division of the truncus medius (+) joins the lateral cord. The median nerve consists of the radix lateralis nervi mediani (*), radix medialis nervi mediani (**) and the anterior division of the truncus medius as an additional branch. The nervus pectoralis medialis originates from the T1 root. **D)** Left BP: there are two thoracicus longus nerves in the BP. One of them, which is marked with a star (*), originates from the C5 root, while the other nerve, which is marked with two stars (**) originates from the C6 and C7 roots.

No anatomical variations were detected at the nervus ulnaris.

Nervus radialis variations were observed in 3 (15%) BPs. This nerve received a thin branch from the anterior division of the TM in one BP (Figure 4), from the MC in the second and from the TI in the third (Figure 2).

In 8 BPs (40%), the nervus medianus was found to be normal, while in 12 BPs (60%), variation was detected. It was observed that the nervus medianus was formed from the anterior division of the TM and MC in two BPs. In 10 BPs (50%), the radix lateralis nervi mediani, radix medialis nervi mediani, and one or more branches from the anterior division of TM were involved in the formation of the nervus medianus, which indicates that it was formed from 3 main roots (Figures 2, 3). In 3 of the 10 BPS, it was observed that the branch from the anterior division of TM merged with the radix medialis nervi mediani in one BP (Figure 2), with radix lateralis nervi mediani in the second (Figure 3), and directly joined the nervus medianus in the third (Figure 2). In 1 of the 10 BPs, the anterior division of TM gave off four branches; one of them joined the

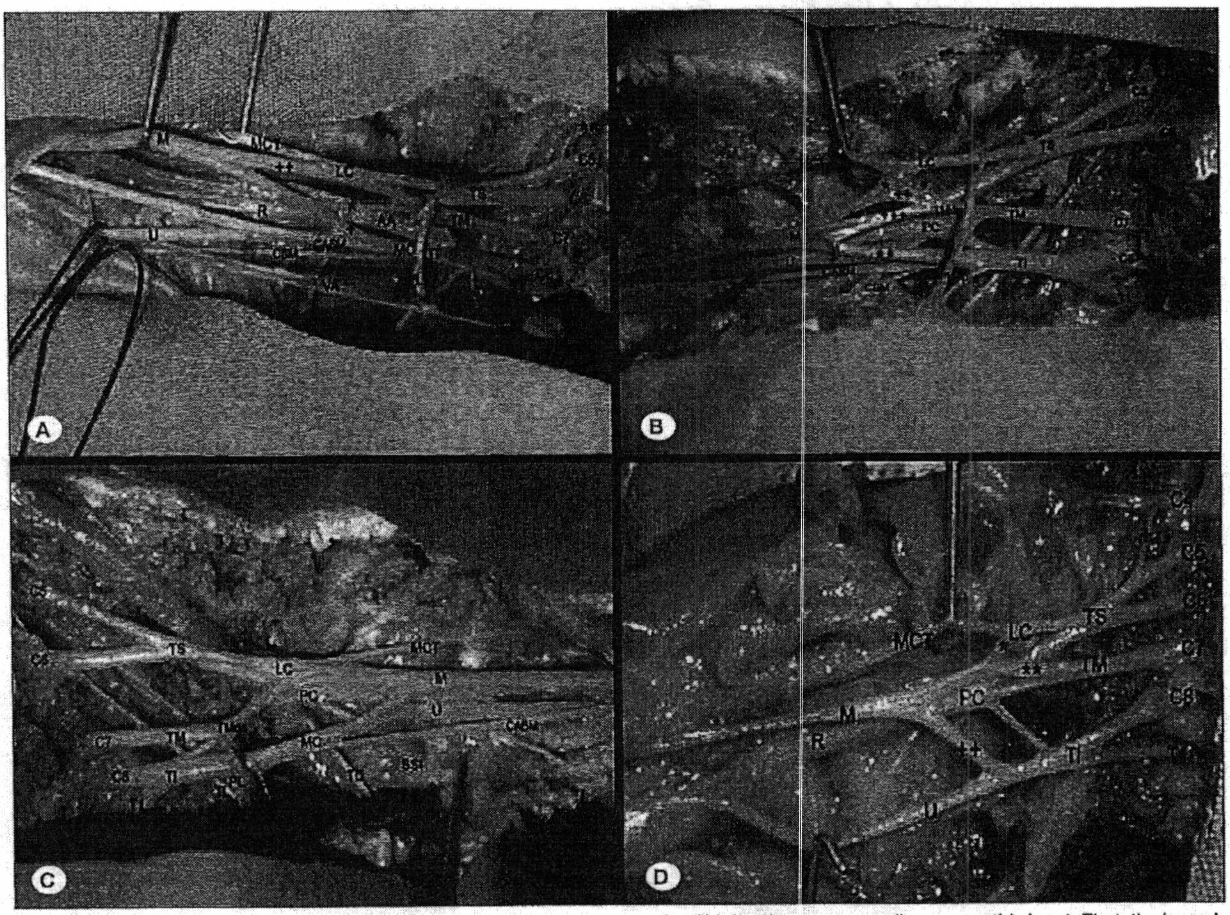


Figure 3: A) Right BP: A branch of the anterior division of the truncus medius (*) joins the nervus medianus as a third root. First, the branch joins the radix medialis nervi mediani (+), then, this conjunction contributes to the median nerve with the radix lateralis nervi mediani (++). The nervus pectoralis medialis originates from the T1 root. The nervus pectoralis lateralis originates from the anterior division of the truncus superior and the truncus medius as a conjunction of those branches. **B)** Right BP: The lateral cord originates only from the anterior division of the truncus superior. There is an additional connection (+) from the anterior division of the truncus medius to the nervus musculocutaneus. The median nerve consists of five roots. Two of the branches (++ and +++) contribute to the nervus medianus together with the radix lateralis (*) and the medialis (**) nervi mediani. The nervus thoracicus longus arises from roots C5 and C6. But, there is no contribution to the nerve from the C7 root. **C)** Right BP: the nervus pectoralis lateralis arises only from the anterior division of the truncus medius. The nervus pectoralis medialis arises from the anterior division of the truncus inferior. The lateral cord joins the anterior division of the truncus medialis to form the nervus medianus. Additionally, the C5 root and the nervus musculocutaneus are thin nerves compared with the normal thicknesses of the nerves. **D)** Right BP: The lateral cord originates only from the anterior division of the truncus superior. A branch of the anterior division of the truncus medius (**) joins the nervus medianus as a third root. First, the branch joins the radix lateralis nervi mediani (*), then, this conjunction contributes to the median nerve with the radix medialis nervi mediani (++).

musculocutaneous nerve, while the others joined the radix lateralis nervi mediani, radix medialis nervi mediani and nervus medianus (Figure 3), In 3 of the 10 BPs, the division anterior of TM divided into two segments as the lateral and medial. The lateral branch combined with the radix lateralis nervi mediani, and the medial branch combined with the radix medialis nervi mediani, and then they formed the nervus medianus (Figures 3, 4).

In 2 BPs (10%), the nervus axillaris originated directly from the posterior division of the TS (Figure 2).

In one BP (5%), the nervus cutaneous brachii medialis and nervus cutaneous antebrachii medialis originated from the T1 root as the main branch, and then divided (Figure 1).

The nervus subscapularis superior and inferior, and nervus thoracodorsalis originated from the posterior division of the TS in one BP (5%). In the other BP (5%), the nervus subscapularis superior and inferior also originated from the posterior division of the TS, while the nervus thoracodorsalis originated from the PC. The nervus subscapularis superior arose from the PC as two branches in one BP (5%). Although the normal sequence of these nerves originating from the PC (proximal to distal)

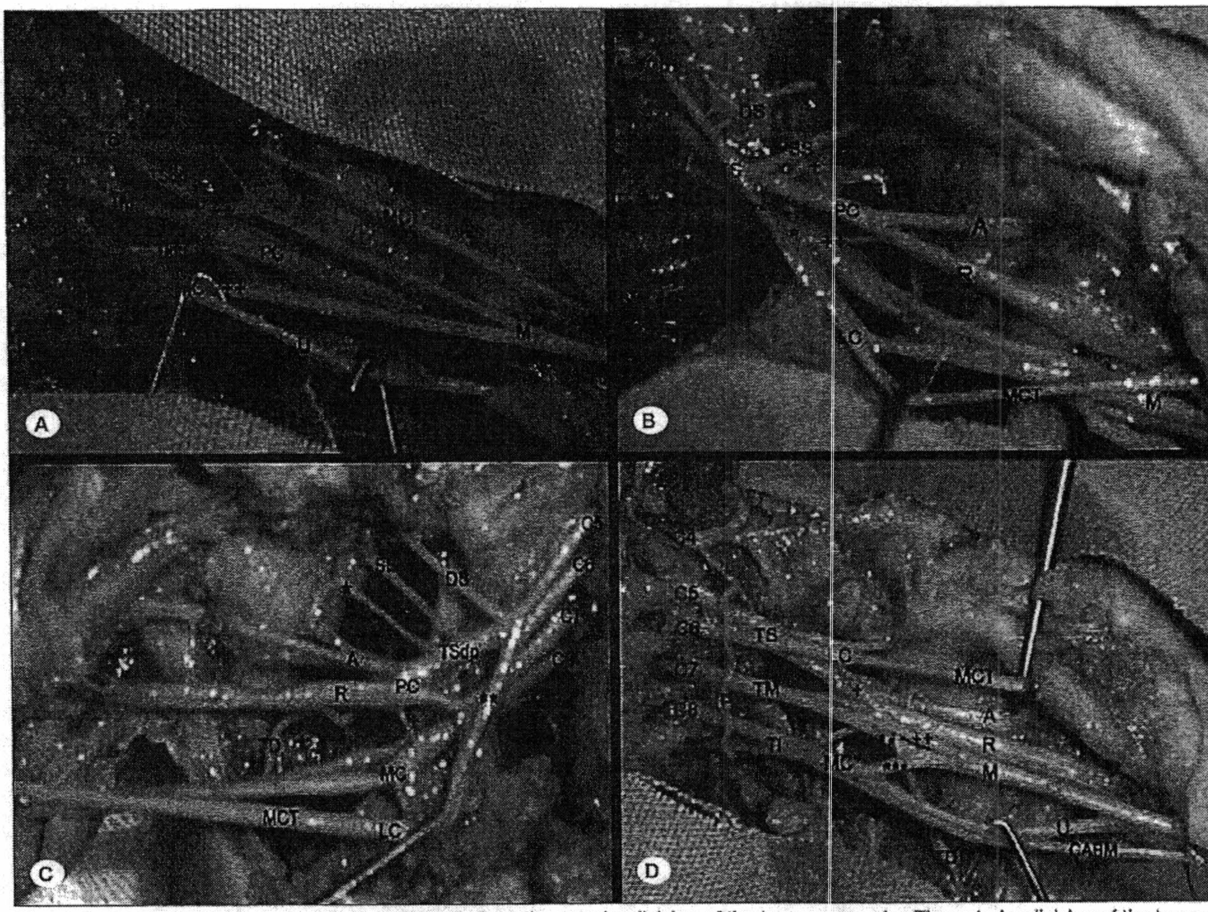


Figure 4: A) Left BP: the lateral cord originates only from the anterior division of the truncus superior. The anterior division of the truncus medius separates into two additional branches. The superior branch (**) joins the radix lateralis nervi mediani (+). The inferior branch (*) joins the radix medialis nervi mediani (++). The nervus medianus consists of the new radixes in the arm level. **B)** Left BP: the lateral cord and the anterior division of the truncus medius were removed with a tool. There is an additional thin connection (++) from the anterior division of the truncus medius (*) to the nervus radialis. The nervus suprascapularis originates from the anterior division of the truncus superior as two branches (SS and *). (**): anterior division of the truncus superior. **C)** Right BP: the lateral cord and the anterior division of the truncus medius were removed with a tool. The nervus suprascapularis originates from the posterior division of the truncus superior as two branches (SS and *). There is an additional thin connection (+) from the medial cord to the nervus radialis (+). (**): anterior division of the truncus superior. **D)** Left BP: the lateral cord originates only from the anterior division of the truncus superior. The nervus medianus consist of four roots. The anterior division of the truncus medius separates into two branches, which are the superior branch (++) and inferior branch (**). The radix lateralis nervi mediani (+) joins the superior branch. The radix medialis nervi median (***) joins the inferior brunch. Then, both conjunctions join each other and the nervus medianus is formed.

is the nervus subscapularis superior, nervus thoracodorsalis, and then the nervus subscapularis inferior, they were arranged in the order of the nervus subscapularis superior, nervus subscapularis inferior and nervus thoracodorsalis in one BP (5%).

The nervus thoracicus longus originated only from the roots C5 and C6 in 2 BPs (10%) (Figure 3). There were 2 nervus thoracicus longus in one BP (5%); one originated only from the C5 root and the other originated from the C6 and C7 roots (Figure 2).

In 13 BPs (65%), the nervus dorsalis scapula originated at the output of the vertebral foramen and the width of the nerve was found to be similar to the branches given to the scalene muscles.

In 7 BPs (35%), the nervus suprascapularis was found to originate proximal to the TS. In 4 BPs (20%), it originated from the posterior division of the truncus superior (Figures 1, 2). There were 2 nervus suprascapularis in two BPs (10%). In one BP, one of the nervus suprascapularis originated from the posterior division of the truncus superior (Figure 4), and the other originated from the anterior division (Figure 4). In the second BP, both nervus suprascapularis originated from the posterior division of the TS.

In 14 BPs, variation was detected in the nervus pectoralis lateralis. In 3 BPs (15%), it originated from the anterior division of the TS, in 4 BPs (20%), it originated from the anterior division of the TM (Figure 3) and in 7 (35%) BPs, it originated from the anterior division of both the TS and the TM (Figures 2, 3).

In one BP (5%), the nervus pectoralis medialis originated from the C8 root, in one BP (5%) it originated from the T1 root (Figure 2) and in 5 BPs (25%), it originated from the anterior division of the TI (Figure 3).

Measurement of Thickness

In only one BP (5%), the C5 nerve root that formed the truncus superior with only the C6 nerve root and without C4 nerve root involvement, was found to be thinner than normal. In the same BP, the nervus musculocutaneous that takes some branches from C5 nerve root was also thin (Figure 3). The measurements of the nerve widths are shown in Table II. The elements of the plexus brachialis were arranged in the following order from large to small according to their average thicknesses: C7>C6>C8>C5 = T1; TS>TI>TM; PC>LC>MC.

DISCUSSION

The normal anatomical structure of BP involves the last four cervical and the T1 spinal nerves. Sometimes, there can be contribution from both the C4 and T2 spinal nerves (20). In this study, it has been found that the BPs of the fetal cadavers were formed by the spinal nerves C4-T2. Kerr classified the prefixed brachial plexus anatomy in three groups with anatomic variations: if there is participation from C4 to C5, it is classified as Group 1 (62.85%); if there is no participation from C4 to C5, it is classified as Group 2 (29.71%); and after a portion of C5 joins C4 and unites the cervical plexus, this group is classified as Group 3 (7.42%) (9). In the present study, 50% of the BPs were found to be prefixed, while 15% of these prefixed BPs were also postfixed. In 10 cases, the BPs were found to have a normal anatomical structure that involved C5-C8 and T1. Kerr Group 3 was not detected in the present study. In a study conducted by Lee et al., a total of 152 BPs were analyzed in 77 adult cadavers and 77% were found to have normal structure (11). Prefixed BPs were found in 21.5% cases and, in only one case, both C4 and T2 were involved. According to the Kerr classification, Lee et al. found that 21.5% of their cases were Group 1, 77% were Group 2 and no cases were in Group 3, which is similar to the results from the present study (11). In another study conducted by Uysal et al., 200 BP were investigated and 25.5% of the cases were found to be in Kerr Group 1, 71.5% had normal structure and 2.5% had postfixed BPs. Kerr Group 3 was not detected in the study (24). A study conducted in Slovakia reported that 48% of the cases had prefixed BP and there was only one case of postfixed BP among 100 BPs (14). Another Brazilian study found that 24% of the cases were prefixed and 4% were postfixed among a total of 54 BPs (4). Finally, a study conducted in Korea reported 77% normal and 21.7% prefixed BPs among a total sample of 152 BPs (23).

Table II: Width of the Structures of Brachial Plexuses

Parameters	Width of nerves (mm)	Mean (mm)
C5 nerve root	0.8-1.1	1.0 ± 0.103
C6 nerve root	1.1-1.4	1.3 ± 0.094
C7 nerve root	1.2-1.5	1.4 ± 0.107
C8 nerve root	0.9-1.2	1.1 ± 0.103
T1 nerve root	0.9-1.1	1.0 ± 0.071
Superior trunk (TS)	1.8-2.2	2.1 ± 0.117
Medial trunk (TM)	1.5-1.9	1.7 ± 0.141
Inferior trunk (TI)	1.7-2.2	2.0 ± 0.152
Lateral cord (LC)	1.7-2.1	1.9 ± 0.138
Medial cord (MC)	1.5-1.8	1.6 ± 0.097
Posterior cord (PC)	2.0-2.4	2.3 ± 0.112

Few studies have been conducted on fetal cadavers. Uzun and Bilgic evaluated 130 BPs from 65 infant cadavers and revealed that 69.23% of the cases had normal origin of BP, while there was a variant connection between the C4 and C5 spinal nerves in 30.77% of the cases. Nonetheless, they found no contribution of the T2 spinal nerve (postfixed) (26). Kerr, Patterson and Scanlon reported the presence of postfixed variation as 30% and 33% in their studies, respectively (9,19). In different studies, a small number of postfixed BP has been reported and no cases were reported in the study by Uzun and Bilgic (26); however, in the present study, postfixed BPs were found in 15% of the cases (4,14,26).

Another study performed by Uysal et al. on 200 BPs from fetal cadavers reported that 71.5% of the cases had normal anatomical structure of the BP. Prefixed BPs were observed in 25.5% of the cases and 2.5% of the cases were found to have postfixed BPs. They found one case in which the C4 and T2 nerves joined the formation. Also, in another case, the TI was formed by the ventral rami of the T1 and T2 nerves (24). In the present study, 3 BPs were found where both the C4 and T2 nerves joined the BP.

In another study conducted by Wozniak et al., spinal nerve root variations were detected in 35 plexuses. In 26 cases, the C4 nerve supernumerary root was found and in two cases the T2 nerve root formed the BP. Additionally, in four plexuses, the T1 nerve root was not observed (27).

The study by Kerr revealed that in 89% of the cases, the superior trunk was formed in the normal anatomical way and in 11% of the cases, variation was found (9). The study by Uysal et al. observed a variation at TS. It was formed by the ventral rami of the C4 and C5 nerves in 1% of cases while in one plexus (24). The study by Nayak et al. described a superior trunk variation that had been formed by the C5, C6 and C7 nerve roots (17). Different authors also found this variation (2,13). Furthermore, the study by Aragão et al. reported that there was a formation of four trunks in 2.5% of the cases (2). In the present study, 10 cases were observed in which the C4, C5, C6 roots formed the TS, while in the remaining 50% of the BPs, the C5 and C6 roots formed the TS.

C7 nerve roots formed all TMs in the present study, similar to Kerr's study (9). No variations were observed in the TM, although the study by Kerr reported that there was a variation of the TM in 6.1% of the cases (9).

The Kerr study determined that the joining of the C8 and T1 formed the inferior trunk in 95% of the cases, and 5% of the cases had variation (9). In 17 BPs (85%), the C8 and T1 roots formed the truncus inferior (TI), while in 3 (15%) BPs, the C8, T1 and T2 roots formed the TI in the present study.

The Kerr study also revealed that no lateral cord was formed in 4% of cases and the MC was formed in all cases. In 36 BPs (20.57%), he was unable to find the real PC unless a single nerve, the nervus radialis, was considered to represent the PC (9). The study by Uysal et al. revealed variations in the LC and MC. The LC consisted of only the anterior division of the TS (2.5%), a branch of the anterior division of the TM joined the MC (5%), a branch of the anterior division of the TM contributed to the LC, and another branch of the anterior division of the TM contributed to the nervus ulnaris (0.5%) (24). In the present study, LCs were formed by only the TS in 7 BPs (35%). In one of the BCs (5%), the anterior division of the TM gave off a small branch to the LC. In one BP (5%), the posterior divisions of the TM and TI first formed the PC, and the posterior division of the TS joined the PC after giving the nervus subscapularis superior and inferior. In the other BP (5%), the posterior division of the TS first gave off the nervus thoracodorsalis, and then formed the PC with the posterior divisions of the TM and TI.

The nervus medianus variants constitute the majority of the anatomical studies of the terminal branches of the plexus brachialis (15,16). The most common variation of the nervus medianus has been reported as the formation from three roots (21,22), similar to the present study (50% of BPs).

The study by Fuss revealed that in 158 BPs, the fibers of the nervus ulnaris originated either from the MC or also from the LC. Lateral root involvement was observed in 56% of cases, which suggested that this should be considered normal and not a variation (5). No anatomical variations and a contribution of the lateral root to the nervus ulnaris were observed in the present study.

In another study conducted by Wozniak et al., distal nerve variations were observed in 35 cases (15.90%)(27). Uysal et al. reported terminal branch variations in 8.5% of the cases they observed where the roots of the nervus medianus joined in the distal part of the arm; in 2.5% of the cases, the axillary nerve was separate from the posterior division of the TS. They also reported a connection in 1% of the cases between the nervus medianus and nervus musculocutaneous (24). In the present study, variations were also observed in the nervus axillaris (10%), nervus radialis (5%), nervus cutaneous brachii medialis and nervus cutaneous antebrachii medialis (5%), nervus thoracicus longus (10%), nervus suprascapularis (35%), nervus pectoralis lateralis (70%) and nervus pectoralis medialis (35%) in the 20 BPs

The study by Kerr revealed that the fibers of the anterior segments of the spinal nerves were counted by microdissection and the thicknesses were determined as C7>C6>C8>C5=T1, according to the number of fibers (9). The study by Uysal et al. reported morphometric measurements of the fetuses. It was determined that C5 and T1 were equal and thin, while C7 and C8 were equal and thick in measurements made at the 3rd trimester. The TM was found to be thinner than the other trunks. The PC was the thickest cord, and the nervus radialis was identified as the thickest terminal branch (24). The study by Uzun and Bilgic revealed the thickness of the plexus brachialis components as C7>C6>C8>C5=T1, TS=TI>TM, PC>LC>MC (26). Since the fetuses were not at the same month, were of different sizes and the development of the fetuses was not completed, it was thought that the lengths and thicknesses of the nerves would not be statistically meaningful. However, the plexus brachialis elements were arranged in the following order from large to small according to their average thicknesses: C7>C6>C8>C5=T1; TS>TI>TM; PC>LC>MC.

CONCLUSION

The plexus brachialis is an important formation due to its complex structure, its relation to other structures, and its anatomical region. This anatomic structure of the plexus must be comprehensively understood by surgeons engaged in brachial plexus surgery.

REFERENCES

1. Akboru IM, Solmaz I, Secer HI, Izci Y, Daneyemez M: The surgical anatomy of the brachial plexus. Turk Neurosurg 20: 142-150, 2010
2. Aragão JA, Melo LO, Barreto ATF, Da Silva Leal AT, Reis FP: Variations in the formation of the trunks of brachial plexus. J Morphol Sci 31:48-50, 2014
3. Dunkerton MC, Boome RS: Stab wounds involving the brachial plexus. J Bone Joint Surg 70-B: 566-570, 1988
4. Fazan VPS, Amadeu AS, Caleffi AL, Filho OAR: Brachial plexus variations in its formation and main branches. Acta Cir Bras 18 Supl 5:14–18, 2003
5. Fuss FK: The lateral root of the ulnar nerve. Acta Anat (Basel) 134:199–205, 1989
6. Gacek RR: Neck dissection injury of a brachial plexus anatomical variant. Arch Otolaryngol Head Neck Surg 116: 356-358, 1990
7. Guday E, Bekele A, Muche A: Anatomical study of prefixed versus postfixed brachial plexuses in adult human cadaver. ANZ J Surg 87:399-403, 2017
8. Hentz VR, Meyer RD: Brachial plexus microsurgery in children. Microsurgery 12:175-185, 1991
9. Kerr AT: The brachial plexus of nerves in man, the variations in its formation and branches. Am J Anat 23:285-392, 1918
10. Laurent JP, Lee R, Shenaq S, Parke JT, Solis IS, Kowalik L: Neurosurgical correction of upper brachial plexus birth injuries. J Neurosurg 179:197-203, 1993
11. Lee HY, Chung IH, Sir WS, Kang HS, Lee HS, Ko JS, Lee MS, Pork SS: Variation of the ventral rami of the brachial plexus. J Korean Med Sci 7:19-24, 1992
12. MacCarty CS: Surgical exposure of the brachial plexus. Surg Neurol 21:593-596, 1984
13. Matejcik V: Aberrant formation and clinical picture of brachial plexus from the point of view of a neurosurgeon. Bratisl Lek Listy 104:291-299, 2003
14. Matejcik V: Variations of nerve roots of the brachial plexus. Bratisl Lek Listy 106:34-36, 2005
15. Nakatani T, Tanaka S: Absence of the musculocutaneous nerve with innervation of coracobrachialis, biceps brachii, brachialis and the lateral border of the forearm by branches from the lateral cord of the brachial plexus. J Anat 16:459-460, 1997
16. Nakatani T, Tanaka S, Mizukami S: Bilateral location of the axillar artery posterior to the medial cord of the brachial plexus. J Anat 16:457-459, 1996
17. Nayak S, Somayaji N, Vollala VR, Reghunthan D, Rodrigues V: A rare variation in the formation of upper trunk of the brachial plexus: A case report. Neuroanatomy 4:37, 38, 2005
18. Ochiai N, Nagano A, Sugioka H, Hara T: Nerve grafting in brachial plexus injuries. J Bone Joint Surg 78-B:754-758, 1996
19. Patterson KW, Scanlon P: An unusual complication of brachial plexus sheath cannulation. Br J Anaesth 65:542-543, 1990
20. Richard LD, Wayne V, Adam WM: Gray's test book of anatomy for student. Elsevier Churchill Livingstone, 2005:661- 663
21. Sargon M, Uslu SS, Celik HH, Aksit D: A variation of the median nerve of the level of brachial plexus in man. Bull Assoc Anat (Nancy) 79:25, 26, 1995
22. Sarsilmaz M, Sendemir E, Celik H, Gumusalan Y, Simsek C: Some variations of the brachial plexus in man. Turk J Med Res 2:161-165, 1993
23. Singhal S, Rao VV, Ravindranath R: Variations in brachial plexus and the relationship of median nerve with the axillary artery: A case report. J Brachial Plex Peripher Nerve Inj 2:21, 2007
24. Uysal II, Seker M, Karabulut AK, Buyukmumcu M, Ziylan T: Brachial plexus variations in human fetuses. Neurosurgery 53: 676-684, 2003
25. Uzmansel D, Kurtoglu Z, Kara A, Ozturk NC: Frequency, anatomical properties and innervation of axillary arch and its relation to the brachial plexus in human fetuses. Surg Radiol Anat 32(9):859-863, 2010
26. Uzun A, Bilgic S: Some variations in the formation of the brachial plexus in infants. Turk J Med Sci 29:573-577, 1999
27. Wozniak J, Kędzia A, Dudek K: Brachial plexus variations during the fetal period. Anat Sci Int 87:223-233, 2012

EXHIBIT 7

Date of Service:	9/5/2014 00:00 PDT
Authored By:	Tse,Raymond W,MD (9/8/2014 12:25 PDT)
Result Status:	Modified
Document Type:	Op Note

Operative Report

Document may Not be Signed/Finalized. See End of report for Electronic Authentication of Signature.

OPERATIVE REPORT

HAMILTON, ZACHARY
JAMES

DOB: 04/09/2014 M -MR #: 01-35-29-63 PATIENT LOCATION: SUR-R5

DATE OF OPERATION: 09/05/2014

PREOPERATIVE DIAGNOSIS:
1. Right brachial plexus palsy (Narakas type IV--flail arm with Horner syndrome).
2. Severe torticollis.
3. Positional plagiocephaly.
4. Shoulder and hand stiffness.

POSTOPERATIVE DIAGNOSIS:
1. Right brachial plexus palsy (Narakas type IV--flail arm with Horner syndrome).
2. Severe torticollis.
3. Positional plagiocephaly.
4. Shoulder and hand stiffness.

OPERATION:
1. Botox chemodenervation of pectoralis major and teres minor.
2. Examination under anesthetic of shoulder, elbow, forearm and hand.
3. Brachial plexus exploration and reconstruction using bilateral sural nerve grafts
A. C5-C8 x2 (3.5 cm).
B. C5-T1 x2 (2.5 cm).
C. C5 to posterior division of upper trunk x1 (4.1 cm).
D. Spinal accessory to suprascapular nerve transfer (performed by Dr. Jeff Friedrich).
E. Cervical plexus nerve transfer to middle trunk.
4. Application of shoulder spica cast stretching fingers and wrist into extension and positioning in supination, elbow flexion and shoulder external rotation with adduction.

SURGEON: Raymond Tse, MD, Attending; Jeffrey Friedrich, MD, Attending; Samuel Lien, MD, Assistant; Rahul Kasukurthi, MD, Assistant; Brinkley Sandvall, MD, Assistant.

ANESTHETIC: General anesthesia.
ESTIMATED BLOOD LOSS: 10 mL.

COMPLICATIONS: None.

FINDINGS: Avulsions of C6, C7, C8 and T1 with a severe scarification of nerve elements intermingled with scarred elements of avulsed muscle.

CLINICAL NOTE: Zachary Hamilton was brought to the operating room today for exploration of his right brachial plexus with reconstruction using nerve grafts and nerve transfers. He had a 10-plexus palsy with flail arm and a Horner syndrome. We were therefore highly suspicious of a complete avulsion of T1. He had no recovery of nerve function and he had changes on CT myelogram and MR myelogram of C6, C7 and C8 root avulsions. This was a severe situation. I talked to the family about different scenarios as far as reconstruction would potentially proceed based upon our exploration today. I explained that there is a good likelihood that there would 4 or more avulsions. If there was good proximal root to graft from, I would target the hand as the priority. I would likely need intercostal nerve transfers to provide nerve input for elbow flexion and I would try to provide shoulder motor stability by transferring spinal accessory nerve to suprascapular nerve. I did discuss the possibility of 5 nerve root avulsions in which case we would need to go to a contralateral C7 nerve transfer to try to obtain axons for reconstruction. I talked about using bilateral sural nerve grafts. We discussed Botox chemodenervation of the internal rotators and exam under anesthetic. Family understood the risks and possible complications of surgery, which include but are not limited to scar, hypertrophic scar, keloid scar, bleeding, infection, nerve injury, vascular injury, tendon injury, lung injury, pneumothorax. Family understood the limitations of what could be achieved surgically. Given the dire situation and the severity of this brachial plexus palsy, we would absolutely not expect any kind of normal function. We would consider reconstruction so that this arm could be a good helper arm as a treatment success, given his situation. He had a flail arm with Horner syndrome suggestive of avulsions and the prognosis generally for this is quite poor. Given his time course without any kind of indication of recovery, this further suggested a poor prognosis. We did talk about the limitations of surgery such that about 80%-90% of children have improvements, whereas 10% have no improvement and 1%-5% have a decrease in function. Zachary does not have much function to begin with and parents wished to proceed with surgery. They understood the additional risks of donor site dysfunction and disability. They understood that we would be sacrificing nerve elements to functioning muscles to innervate the brachial plexus. All wished to proceed.

OPERATIVE NOTE:
I examined Zach's limb while he was under anaesthetic. His arm had lots of stiffness that was consistent with chronic brachial plexus palsy and no stretching, therapy, or range of motion exercises. Dr. Steinman examined him with me and confirmed the same. It was difficult to place fingers and wrist into extension due to tight finger flexors and it was difficult to externally rotate his shoulder because of generalized shoulder stiffness. His pectoralis major and teres minor seemed particularly tight. We therefore performed chemodenervation of both of these muscles using botulinum toxin placing 28 units and 14 units in each respectively.

Patient was positioned supine on the operating room table. We did position the baby carefully placing a gel ramp between his scapulae and positioning his head on a Z-Flo pillow. We changed his head position and his ankle position at approximately every 3 hours to avoid any pressure ulceration. We did a 2-team approach with one team exploring the brachial plexus and the other team harvesting sural nerve grafts.

Up in the head and neck region, the head was positioned facing away from the affected side with the chin to shoulder. The right neck, shoulder, and ear were prepped and draped in appropriate sterile fashion. I designed an incision along the lower neck crease with an extension along the posterior border of the sternocleidomastoid muscle. Here, I incised sharply through skin using a #15 blade and used monopolar cautery to dissect through subcutaneous fat and platysma muscle. The flap was raised based superolaterally. The flap was elevated in a subplatysmal plane. The cervical plexus was identified and each of the branches was traced back to the C4 root. It is here that the dissection proceeded medially such that we could identify the phrenic nerve. This nerve responded in a positive manner to electrical stimulation, thereby confirming that this was the phrenic nerve. I incised the supraclavicular fat pad along the posterior border of the sternocleidomastoid muscle as well as along the superior border of the clavicle. The fat pad was elevated based superolaterally to expose a large mass of scarification. It was very difficult to recognize any kind of nerve structures, given that everything was severely scarified and there was torn muscle intermingled with a ruptured nerve. I did take quite some time to dissect out the brachial plexus. This was extremely tedious given the severe scarification of structures, and once again intermingling of tore nerve elements with torn muscle elements. I eventually found a healthy proximal C5 root. I could see that C6 had been completely ruptured, at least at the level of the intervertebral foramen. There was a small wisp of scar that I could follow into the foramen and I sent this off to Pathology. They confirmed that there is no real usable nerve architecture here for grafting. Distally, I transected the suprascapular nerve, posterior division of upper trunk and anterior division of upper trunk beyond what I could see as the neuroma. The frozen section pathology reported extremely poor nerve architecture for the suprascapular nerve and it took an additional 2 cuts in this nerve further distal before we could obtain reasonable nerve architecture. I could not trim the suprascapular nerve any further distal with our exposure as this was quite distant into the soft tissues. Similarly, I had to trim the posterior division of upper trunk further distally and the C5 root more proximally in order to obtain acceptable nerve architecture. Once again, I was working in the retroclavicular space and posterior division of upper trunk was trimmed as distally as it could be accessed. I did find a very scarified middle trunk with progressively greater scarification towards the C7 foramen. In fact, the root looked avulsed as there is only a thin spindle of scar emanating from the C7 foramen. I transected the middle trunk where I thought that things will be healthy and submitted this to Pathology.

I next found the lower trunk. The convergence of C8 and T1 was extremely distal and in the retroclavicular region, suggestive of a more proximal avulsion-type of scenario. C8 was somewhat scarified proximally and again the root petered out to nothing as we approached the foramen. The T1 root was similar. Once again, there were empty foramens for C6, C7, C8 and T1. These all confirmed avulsion of these roots. I did stimulate each of the proximal roots. With 1.5 mV at C5 I got 2+ pectoralis. With 2.0 mV at C6 I got nothing. With 1.5 mV at C7 and I did get a 2+ response of the pectoralis major and with 1.5 mV at C8 I did get a 1+ response at the pectoralis major and 1+ response for wrist flexion.

Dr. Friedrich became involved as the second surgeon, given the severe scarification and the extreme complexity of this exploration. Once again, everything was severely scarred down and the dissection was difficult and tedious.

With a single C5 root, the primary target would be to innervate the hand. I therefore designed a reconstruction which would use 4 cable grafts to C8 and T1 of the lower trunk. I would use the sural nerve graft for this. The distance from C5 to each of the C8 and T1 recipients was about 3.5 cm. From C5 to posterior division of upper trunk it was about 4.1 cm. I would use spinal accessory nerve to suprascapular nerve and the cervical plexus to middle trunk for reinnervation of these. I would leave the anterior division of upper trunk unsatisfied with the plan to return for intercostal to musculocutaneous nerve transfer to try to achieve some elbow flexion. I decided that it would not be ideal to perform the intercostal nerve to musculocutaneous nerve transfer today given that the exploration and dissection of the brachial plexus took quite a long time and we would be starting intercostal to musculocutaneous nerve transfer quite late in the day. There is also an advantage to doing this another day, given that it would be preferable to cast Zach in external rotation to try to stretch the shoulder as well as to cast him with some wrist and finger extension. If I was to perform intercostal nerve transfer to musculocutaneous nerve, the preference would be for a cuff and collar sling with internal rotation and adduction, which would be counter to the stretches that we would be trying to achieve. Zach was very stiff and it was quite apparent that he had had no real therapy or stretching exercises.

Prior to nerve exploration, I did inject the pectoralis major with 4 units/kg of botulinum toxin. I also injected the teres minor and given that this was a smaller muscle I used 2 units/kg for this muscle injection. Prior to beginning, I did find that everything was very stiff. I could only externally rotate the shoulder to about 30 degrees shy of neutral. With the slow gentle and progressive stretch, I could get him out to about 60-75 degrees of external rotation beyond neutral. Similarly, his finger and wrist flexors were tight and it was difficult to hold them in full wrist extension with full finger extension.

Our second team was working down in the legs. Bilateral sural nerve grafts were harvested. This was done through 3 incisions. The first was in the popliteal fossa. The second was along the lateral malleolus through one of the ankle creases. The third was via a short 2 cm transverse incision at the junction of the tendinous and muscular part of the gastrocnemius muscle.

Here, we started by incising sharply through skin and then bluntly dissecting through subcutaneous tissues. The sural nerve was identified and followed proximally. It was unroofed along its length, opening the fascia above it. The nerve was followed into the popliteal fossa and beyond the popliteal fossa. The sural nerve was then lysed from the main nerve for several centimeters beyond the popliteal crease. We then dissected circumferentially around the nerve and would then transect the nerve as far proximal as possible. This proximal end was marked so that the nerves could be reversed for nerve grafting. The nerve was withdrawn from each of the more distal incisions and then finally harvested from the ankle. The excess fat was removed from the nerve. The nerve grafts were treated extremely gently throughout. The legs were closed in several layers using 4-0 Vicryl in the deep soft tissues and deep dermis. We used 5-0 Monocryl running intracuticular in the skin. This was reinforced with Dermabond glue. This was covered with Tegaderm occlusive dressings. We did use some Coban in each of the legs. Both legs were harvested. All this was done in a sterile manner.

Up in the head and neck region, the nerve grafts were trimmed to appropriate length. We then swung in the microscope and secured the nerve grafts to the proximal C5 stump. I used fibrin glue to do so. T1 grafts would be positioned posteriorly and inferiorly. C8 grafts would be secured superiorly. The posterior division of upper trunk graft would be positioned anteriorly. Each of the nerve grafts was glued in proximally using Tisseel fibrin glue. The microscope was then swung 90 degrees and each of the grafts was secured to the distal targets, ensuring that there was good nerve-to-nerve contact with the recipient. I used a single graft to the posterior division of upper trunk. In order to provide sensation of the hand, I used the cervical plexus that I had transected distally previously. Each of the 3 large branches was coapted to the middle trunk using Tisseel glue. I had to use a short segment of sural nerve as a graft for one of these cervical plexus branches.

Prior to the nerve graft reconstruction and nerve transfer reconstruction, we did harvest the spinal accessory nerve. This was performed by Dr. Friedrich. He left several branches intact to the trapezius muscle proximally and then dissected this spinal accessory nerve as far distal down the back as possible. The motor function was confirmed by electrical stimulation. The nerve was transected and then tunneled through the supraclavicular fat pad for coaptation to the suprascapular nerve. Each of the distal nerve coaptations was performed with Tisseel glue.

EXHIBIT 8

Anat Sci Int (2012) 87:223–233
DOI 10.1007/s12565-012-0150-1

ORIGINAL ARTICLE

Brachial plexus variations during the fetal period

Jowita Woźniak · Alicja Kędzia · Krzysztof Dudek

Received: 20 January 2012 / Accepted: 7 August 2012 / Published online: 4 September 2012

Abstract The brachial plexus is an important nervous system structure. It can be injured during the perinatal period and by postnatal damage. The goal of this study was to assess human fetal brachial plexus variability. A total of 220 brachial plexuses were surgically prepared from 110 human fetuses aged 14–32 weeks of fetal life (50 females and 60 males) ranging in CRL from 80 to 233 mm. The study incorporated the following methods: dissectional and anthropological, digital image acquisition, digital image processing using Image J and GIMP software, and statistical methods (Statistica 9.0). Symmetry and sexual dimorphism were examined. Anomalies of the brachial plexuses were observed in 117 (53.18 %) cases. No sexual dimorphism was found. It was observed that cord variations occurred more often on the left side. Division variants (33.64 %) occurred most often, but also cords (18.18 %) as well as root nerves and terminal ramifications (15.90 %) were found. Trunk anomalies were rare and occurred in only 5.45 % of plexuses. Three height types of median nerve roots in combination with the nerve were distinguished. In one-third of cases, median nerve root connections were found below the axillary fossa and even half in the proximal part of the humerus. In conclusion, the brachial plexus was characterized for anatomical structural variability. Most often division and cord variations were observed. Anomalies occurred regardless of sex or body side except for cord variants. Brachial plexus variation recognition is significant from the neurosurgical and traumatological point of view.

Keywords Brachial plexus · Morphology · Variations · Human fetuses

Introduction

The fetal period is extremely important in human development. In the course of differentiation, five cellular groups are distinguished: epithelial, muscular, nervous system with gray matter and blood as well as connective tissues.

The available literature does not provide much information concerning nerve and limb development in fetal life. The embryonic period was observed by Lewis (1902), Shinohara et al. (1990), and Rodriguez-Niedenführ et al. (2001, 2003). Lewis (1902) described the embryonic development of the human brachium. The first differentiation of the mesenchyma took place at about the 4th developmental week, and in this period C_4, C_5, C_6, C_7, C_8, and Th_1 brachial plexus spinal nerve continuity was noted. Only three outlines of nerve cords could be found in this plexus. The enormous size of the cervical inferior nerves was significant at about the 5th week. The C_4 nerve root did not form the plexus. The nervous plate made from five spinal nerves underwent clear branching into ventral and dorsal branches. The author observed germ formation of the following nerves: suprascapular, lower and upper subscapular, musculocutaneous, median, ulnar, and long thoracic. In the 6th week, the nerves migrated in a caudal direction. In the 7th week, the brachial plexus posterior

J. Woźniak (✉) · A. Kędzia
Department of Normal Anatomy, Medical University of Wroclaw, Chalubinskiego 6a, 50-368 Wroclaw, Poland
e-mail: jowita_wozniak@yahoo.com

A. Kędzia
e-mail: kedzia.alicja@gmail.com

K. Dudek
Institute of Machines Design and Operation, Technical University of Wroclaw, Wroclaw, Poland

inclination was dominant. The plexus had already been formed to the first rib height, and the three newly formed cords could not be separated yet. Shinohara et al. (1990) examined upper limb nerve development on material consisting of eight human embryos (13–21 as per Carnegie, 32–52 days of fetal life). On the 32nd day, C_5–Th_1 nerve elongations were formed, and on the 33rd day, the nerves united, starting brachial plexus formation. On days 39–40, the median, radial, and ulnar nerves reached the palm level. On days 49–50, brachial plexus orientation and structure were similar to those observed in adults.

Rodriguez-Niedenführ et al. (2001, 2003) examined the embryonic development of upper limb vascularization and described arterial variants. The tests were on 112 embryos in developmental stages: 12–23 (crown-rump length (CRL): 3.5–30 mm). At stage 15, the subclavicular and axillary arteries were distinguished; at stage 17, the brachial artery; at stage 18, the antebrachial arteries were separated, and at stage 21, they reached their final morphological form. In turn, Castellana and Kosa (1999), observed cervical vertebral development in fetuses and newborn babies. The authors found that in the 4th–10th month period, it is vertebral size not shape that undergoes changes.

Uysal et al. (2003) are among the few research workers who carried out surveys of the fetal brachial plexus. A total of 200 brachial plexuses (13–40 weeks of fetal life) were observed, and their morphology was checked. In the 13th week, the plexus was found to be thoroughly formed. The fetal period was the focus of the studies carried out by Uzmansel et al. (2010). The available literature contains a small number of examinations of brachial plexus morphology in the fetal period, so filling this subject gap was the aim of the observations.

The goal of this article was to present brachial plexus variations in human fetuses.

Materials and methods

The material consisted of 220 brachial plexuses originating from 110 fetuses aged 14–32 weeks of fetal life in the CRL range of 80–233 mm, including 50 females and 60 males. All of the material belonged to the Normal Anatomy Department, the Medical University of Wroclaw. Fetuses that did not reveal symptoms of postmortum autolysis or external developmental defects were incorporated in the studies. The fetuses were preserved in formaldehyde solution and maintained for the study period.

The survey methodology included: anthropological and dissection, image digital acquisition, Image J—computer transformation system, GIMP software, and statistical methods (Statistica software package). The anthropological method was based on fetal age assessment with the use of CRL size in conjunction with the Scammon-Calkins formula (Carlson 1999). Microsurgical instruments supported with optical microscopes were used during dissection procedures. The combination of classical methods (dissectional and anthropological) and a contemporary image computer transformation system (not used in brachial plexus studies before) was an innovation. Image J is a free software (available on the manufacturer's web-site: http://www.rsb.info.nih.gov/ij), which includes elaborated functions of image processing or structural 3D visualizations supporting the morphological assessment. The GIMP software contains numerous functions operating the graphics. The use of digital image acquisition as well as the Image J system prevented survey material damage. A 0/1 system was used to evaluate variants of the particular plexus parts: 0, normal anatomical structure; 1, presence of anomalies. This method was used to define the typology of the median nerve into nerve fusion height: 0, nerve fusion in the axillary fossa; 1, fusion in half of the humeral proximal length; 2, fusion in half of the humeral distal length.

Due to the presence of anomalies, which were co-developed by more than one part of the brachial plexus, indices of variability of the brachial plexus were obtained from a larger number of features.

Symmetry and sexual dimorphism were examined by contingency tables as well as chi-square tests or Fisher's test (when any subgroup number was smaller than 6).

Results

No statistically significant difference ($P > 0.05$) in morphological variant prevalence was found between male and female fetuses (Table 1). Table 2 presents the collective results of the qualitative feature observations of the left and right side. Cord variants occurred more often on the left. Generally, 117 (53.18 %) of the examined plexuses were characteristic for their distinct anatomical structure. In the remaining cases, the brachial plexus had a typical anatomical structure. Spinal nerve roots from C_5 to T_1 formed the proper plexus; nerve roots C_5–C_6 formed the upper trunk; C_7 formed the middle trunk, whereas nerve roots C_8–T_1 formed the lower trunk. From C_4 to C_5, the nerve roots created the dorsal scapular nerve, from C_5 to C_7 the long thoracic nerve as well as from the upper trunk nerve to the subclavius and suprascapular nerve. Each of the trunks divided into two divisions—anterior and posterior. Posterior divisions merged together into the posterior cord from which the following kinds of nerves led: the long radial nerve as well as the short axillary nerve, the lower and upper subscapular nerve, and the thoracodorsal nerve. Anterior divisions of the middle and upper trunk created the lateral cord, which branched into the long nerves—the musculocutaneous and lateral root of the median nerve and

Table 1 Number (percentage) of observations of qualitative brachial plexus morphology features on the left and right sides in sex subgroups; comparison results

	Left side (%)			Right side (%)		
	F $N = 50$	M $N = 60$	P	F $N = 50$	M $N = 60$	P
Plexus structure type						
0, normal	25 (50)	24 (40)	0.293	23 (46)	31 (52)	0.554
1, morphological variant	25 (50)	36 (60)		27 (54)	29 (48)	
Nerve root structure type						
0, Normal	42 (84)	52 (87)	0.693	40 (80)	51 (85)	0.491
1, Morphological variant	8 (16)	8 (13)		10 (20)	9 (15)	
Trunk structure type:						
0, Normal	46 (92)	55 (92)	0.615	47 (94)	60 (100)	0.091
1, Morphological variant	4 (8)	5 (8)		3 (6)	0 (0)	
Division structure type						
0, Normal	33 (66)	41 (68)	0.795	32 (64)	40 (67)	0.770
1, Morphological variant	17 (34)	19 (32)		18 (36)	20 (33)	
Cord structure type						
0, Normal	41 (82)	43 (72)	0.296	42 (84)	54 (90)	0.514
1, Morphological variant	9 (18)	17 (28)		8 (16)	6 (10)	
Terminal nerves structure type						
0, Normal	42 (84)	52 (87)	0.902	40 (80)	51 (85)	0.662
1, Morphological variant	8 (16)	8 (13)		10 (20)	9 (15)	
Median nerve connection type						
0, In axillary fossa	30 (60)	36 (60)	0.540	35 (70)	45 (75)	0.698
1, At ½ of the humeral proximal length	20 (40)	23 (38)		13 (26)	14 (23)	
2, At ½ of the humeral distal length	0 (0)	1 (2)		2 (4)	1 (2)	

short lateral pectoral nerve. The anterior division of the lower trunk passed into the medial cord, which created long nerves: the brachium and antebrachium medial cutaneous nerves, ulnar nerves, and medial root of the median nerve, which together with the lateral root created the median nerve and short median pectoral nerve. In ours study, the most often observed were division variants (33.64 %)—every third plexus, cord variant (18.18 %), nerve roots, and final nerves (15.90 %). Trunk anomalies were found sporadically (5.45 %).

Three morphological types of median nerve root connection height were distinguished: type I (normal) in which the roots fused into the nerve in the axillary fossa, type II in which the roots fused below the axillary fossa at half of the humeral proximal length, and type III where the fusion was found at half of the humeral distal length. Figure 1 presents these types, and it is clear that type II was observed significantly more often on the left side (Table 2).

Nerve root anomalies

Spinal nerve root variants were detected in 35 plexuses. In 26 cases, the C_4 nerve supernumerary root was found, and in 10 plexuses this anomaly was bilateral. In two cases the Th_2 nerve root formed the brachial plexus. In addition, the Th_1 nerve root was not seen in four plexuses, a C_6–C_7 nerve supernumerary connection was found in one case (Fig. 2), and C_7–C_8 nerve supernumerary connections were detected in two plexuses (Fig. 3).

Trunk anomalies

Trunk anomalies were found in 12 cases (5.45 %). The absence of the typical upper trunk was observed in three plexuses, and the C_4 nerve supernumerary root formed these anomalies as well. In two of them, a separate trunk was constituted by C_4 as well as C_5 nerve roots. The trunk shaped two branches (composing lateral and posterior cords), and the C_6 root formed a separate conjunction with the lateral and posterior cords. In another case (Fig. 4), C_4 and C_5 roots constituted individual binding and divided into two branches. The two branches merged with the C_6 nerve ramification to form anterior and posterior divisions joining the lateral and posterior cords. In four cases, the middle trunk was formed by C_7 and C_8 nerve roots, and simultaneously, the lower trunk was made of the Th_1 nerve root only. In a single case, the authors observed some

Table 2 Number (percentage) of observations of fetal brachial plexus morphology qualitative features in the side subgroups; comparison results

Qualitative feature	Body side		
	L (%) $N = 110$	R (%) $N = 110$	*P*
Plexus structure type			
0, Normal	49 (45)	54 (49)	0.499
1, Morphological variant	61 (55)	56 (51)	
Nerve root structure type			
0, Normal	94 (85)	91 (83)	0.580
1, Morphological variant	16 (15)	19 (17)	
Trunk structure type			
0, Normal	101 (92)	107 (97)	0.067
1, Morphological variant	9 (8)	3 (3)	
Divisions structure type			
0, Normal	74 (67)	72 (65)	0.775
1, Morphological variant	36 (33)	38 (35)	
Cord structure type			
0, Normal	84 (76)	96 (86)	**0.035**
1, Morphological variant	26 (24)	14 (14)	
Terminal nerve structure type			
0, Normal	94 (85)	91 (83)	0.580
1, Morphological variant	16 (15)	19 (17)	
Median nerve connection type			
0, In axillary fossa	66 (60)	80 (73)	**0.048**
1, At ½ of the humeral proximal length	43 (39)	27 (25)	
2, At ½ of the humeral distal length	1 (1)	3 (3)	

Bold values indicate statistically significant results ($P < 0.05$)

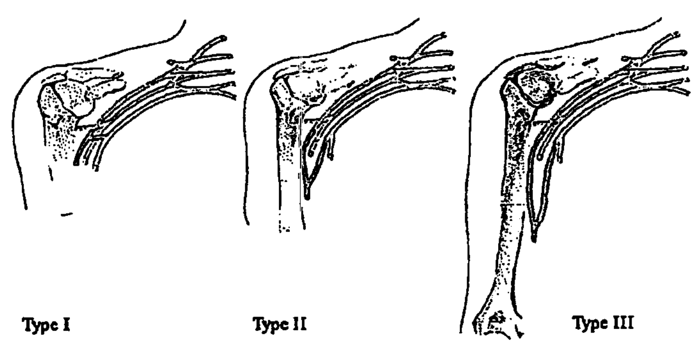


Fig. 1 Median nerve root connection types in relation to the axillary fossa and humeral length: type I, nerve roots combine into a nerve in the axillary fossa; type II; nerve roots combine into the median nerve at half of the humeral proximal length; type III, nerve roots combine into the median nerve at half of the humeral distal length

accessory connections of the middle trunk with anterior divisions of the upper trunk. In three plexuses, the lower trunk formed a supernumerary connection with the lateral cord. The anomalies were accompanied by variability in the form of additional connections between the lateral cord and medial root of the median nerve (Fig. 5).

Division anomalies

Division anomalies were observed in 74 plexuses (33.63 %). Divisions of upper trunk variants were observed in a single case, and it was a supernumerary connection between the anterior division of the upper trunk and anterior division of the middle trunk (ADMT). The most frequent variability concerned the ADMT and was observed in 63 cases. ADMT absence was found in one plexus (Fig. 6). In two cases this division connected with the medial cord. Figure 7 presents the most common anatomical variants of the ADMT. Figure 7a shows ADMT binding with the lateral root of the median nerve, which was observed in three cases. Figure 7b presents the ADMT's direct connection with the median nerve, which was observed in four plexuses. Figure 7c demonstrates

ADMT connection with the medial root of the median nerve in one plexus. In Fig. 7d, the ADMT gives off supernumerary fibers to the median nerve (1 case). In Fig. 7e, the ADMT forms the lateral root of the median nerve, not joining the lateral cord, which is formed only by the anterior division of upper trunk (Fig. 8); this was found in four plexuses. Figure 7f is similar to 7e, but the division gives off an additional connection with the medial root of the median nerve (1 case). In Fig. 7g, in three cases, the ADMT joined the lateral cord and gave off two extra parts to the medial root and lateral root of the median nerve. In turn, in ten cases, the ADMT bifurcated, joining both roots of the median nerve, which is illustrated at point h (Fig. 7h). Type I was observed most often, and it manifested additional fusion of the ADMT and medial root of the median nerve, which was observed in 32 plexuses (Figs. 7i, 9).

However, in one plexus, the ADMT was found to give off extra fibers to the double lateral root of the median

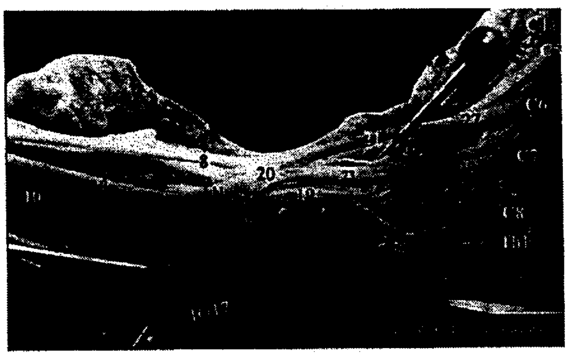


Fig. 2 Right body site: C_6 nerve root and C_7 nerve root connection (*red stars*), C_4 nerve root present (*blue stars*), as well as additional lateral root of median nerve (*green stars*), where: C_5–Th_1 brachial plexus roots, *8* musculocutaneous nerve, *9* axillary nerve, *10* radial nerve, *11* lateral root of the median nerve, *12* medial root of the median nerve, *13* median nerve, *14* ulnar nerve, *16/17* brachium and antebrachium medial cutaneous nerves, *18* medial cord, *19* posterior cord, *20* lateral cord, *21* anterior division of upper trunk, *22* posterior division of upper trunk, *23* anterior division of middle trunk (ADMT), *24* posterior division of the middle trunk, *25* posterior division of lower trunk, *26* anterior division of lower trunk, *27* upper trunk, *28* middle trunk, *29* lower trunk

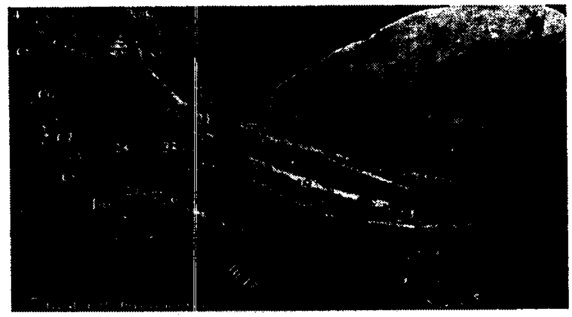


Fig. 4 Left body site: no typical upper trunk, C_4 (*red stars*) and C_5 roots form individual connection dividing into two branches that join the C_6 nerve root ramification (*blue stars*) to create anterior (*21*) and posterior (*22*) divisions as well as anterior division of the middle trunk (*23*) and additional anomalies (*green stars*)

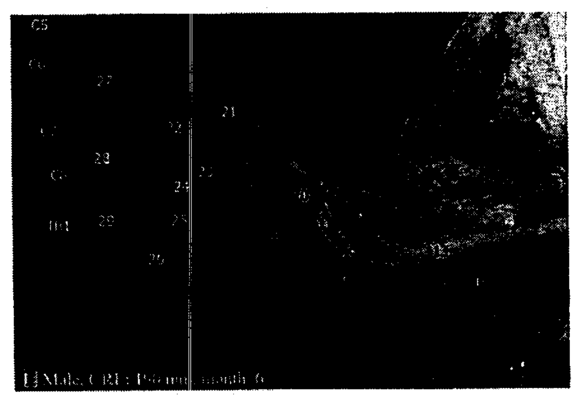


Fig. 5 Left body site: coexistence of accessory division of lower trunk (*29*, *red stars*) with median nerve double lateral root—*green stars*

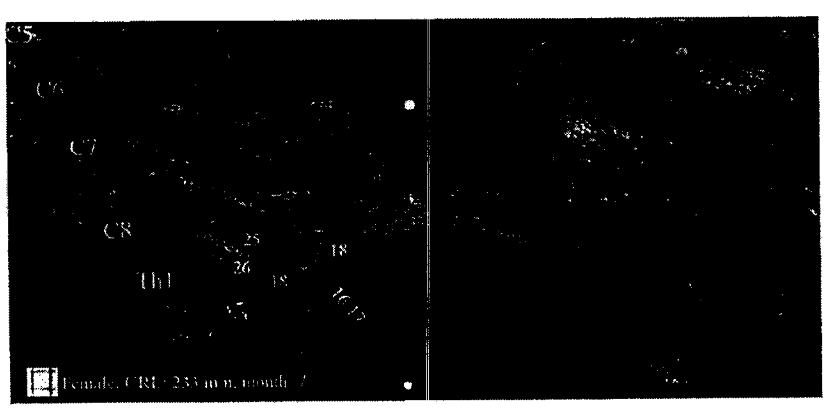


Fig. 3 Left body site: anterior division of the middle trunk (ADMT)—*23* reaches medial cord—*18*. Median nerve goes off lateral cord-nerve roots, poorly shaped. C_7 nerve fibers combine C_8 nerve fibers (*red stars*)

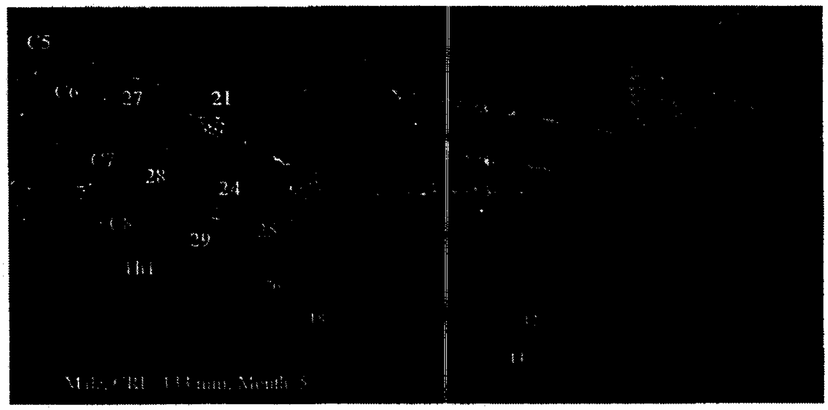


Fig. 6 Left body site: lack of anterior division of the middle trunk (ADMT; *23*), lack of the typical lateral cord, anterior division of the upper trunk (*21*) forms the musculocutaneous nerve (*8*) and lateral root of the median nerve (*11*), posterior cord (*19*) gives off accessory fibers to the lateral (*11*; *blue stars*) and medial (*12*) roots (*red stars*) of the median nerve (*13*)

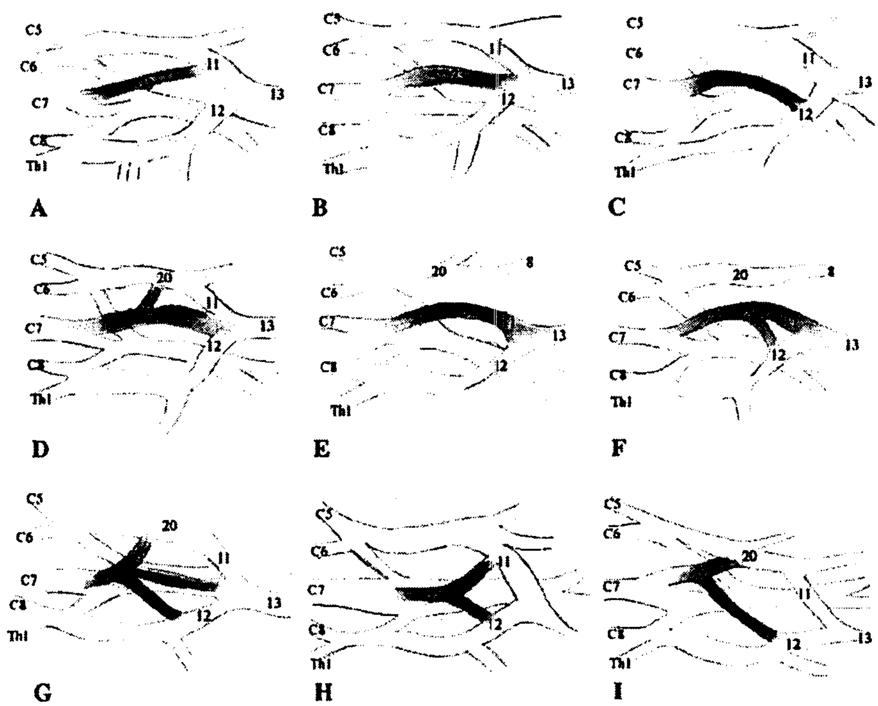


Fig. 7 Median nerve root variants and anterior division of the middle trunk (ADMT) variants, where: C_5–Th_1 brachial plexus roots, *8* musculocutaneous nerve, *11* lateral root of the median nerve, *12* medial root of the median nerve, *13* median nerve, *20* lateral cord, *23* anterior division of the middle trunk (ADMT)

nerve formed from the lateral cord fibers. Posterior division of middle trunk anomalies were detected in two cases only. In the first case, the anomaly was a division and medial cord additional connection. In the other case, the anomaly coexisted with a posterior division of the lower trunk, and both divisions gave off accessory fibers, which combined to form the common trunk, but after division, they joined median nerve roots (Fig. 10). In turn, in eight plexuses, posterior division of lower trunk anomalies occurred. In four of the cases, the division joined the radial nerve, and the posterior cord was formed by posterior divisions in the middle and upper trunks (Fig. 11). In the remaining four cases, this branch went off the C_8 nerve and not the lower trunk.

Cord anomalies

Cord variations were observed in 40 cases, being second in respect to the frequency of anomaly detection in 18.18 % of examined plexuses.

The lateral cord being formed by anterior division of the upper trunk was only found in eight plexuses (Fig. 7a–c).

Fig. 8 Right body site: no lateral cord. Anterior division of the middle trunk (ADMT) (*23*) forms the lateral root of the median nerve (*11*). The musculocutaneous nerve (*8*) constitutes prolongation of the anterior division (*21*) of the upper trunk (*27*)

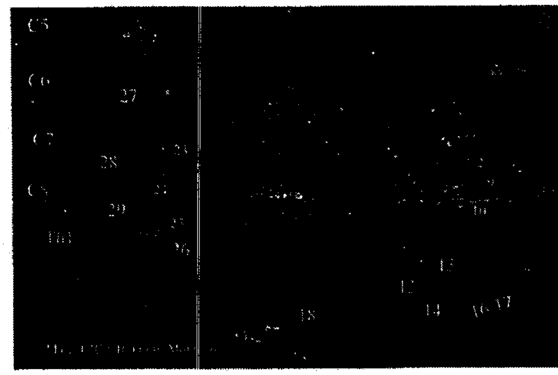
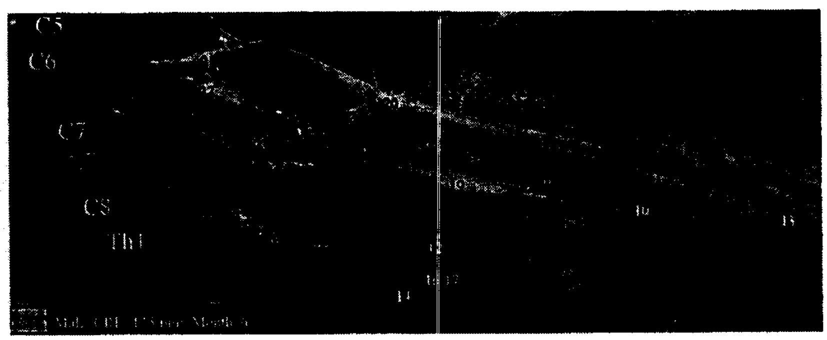


Fig. 10 Left body site: posterior division of the middle trunk (*24*; *red stars*) along with posterior division of the lower trunk (*25*; *green stars*) combines to form the "stem", branches (*blue stars*), and joins the median nerve roots

Fig. 9 Left body site: anterior division of the middle trunk (ADMT) (*23*) gives off accessory fibers (*red stars*) to the medial root (*12*) of the median nerve



In turn, the variation devoid of the lateral cord proper but characteristic for anterior division of the upper trunk running into musculocutaneous nerve was observed in five cases (Figs. 7e, f, 8). In one plexus, the lateral cord was found to be supplied with fibers coming directly from the C_4 nerve root. Additional connection between the cord and medial root of the median nerve was the most frequent anomaly of the lateral cord. It was observed in 20 plexuses and resembled the median nerve *double lateral root*. The median nerve *triple lateral root* in the form of an accessory connection between the lateral cord and medial root of the median nerve was observed in one plexus (Fig. 12). Posterior cord variants were seen in five cases. In four plexuses (Fig. 11) the cord was built of posterior divisions of the middle and upper trunks. Posterior division reached the radial nerve. In one case, two divisions went off the cord, and they combined with the medial and lateral roots of the median nerve (Fig. 6). Medial cord variations were not observed.

Distal ramification anomalies

Brachial plexus distal nerve variations were observed in 35 cases, which constituted 15.90 % of all examined plexuses. Musculocutaneous nerve variants were seen in 23 observations, and in 5 cases, the nerve was formed of anterior division of the upper trunk extension (Figs. 7e, f, 8). In 16 cases, the nerve was the anterior division of the upper trunk ramification before the division's fusion with the ADMT. In one plexus, anastomosis between musculocutaneous and median nerves was observed, and another case presented this relation with the radial nerve (Fig. 13). Median nerve roots revealed significant variability, and these variants are presented in Fig. 7.

Nerve root fusion into the median nerve underwent changes in relation to humeral length. The median nerve went off the lateral cord in six plexuses. Besides, axillary nerve anomalies were observed as the nerve descended from the posterior division of the upper trunk in four cases. In two cases, the medial brachial and antebrachial cutaneous nerves branched directly from the Th_1 nerve root.

Discussion

In the examined material (220 plexuses), 117 plexuses revealed nontypical anatomical structure, and the majority

Fig. 11 Right body site: connection between the posterior division of the lower trunk (25) and radial nerve (*10*; *red star*), C_4 root present, posterior cord (*19*) formed by posterior division of the upper trunk (22) and posterior division of the middle trunk (*24*)

Fig. 12 Right body site: nerve double fibers (*red stars*) going off the lateral cord (*20*) to the medial root (*12*) of the median nerve (*13*) as well as the C_4 root are present (*white stars*)

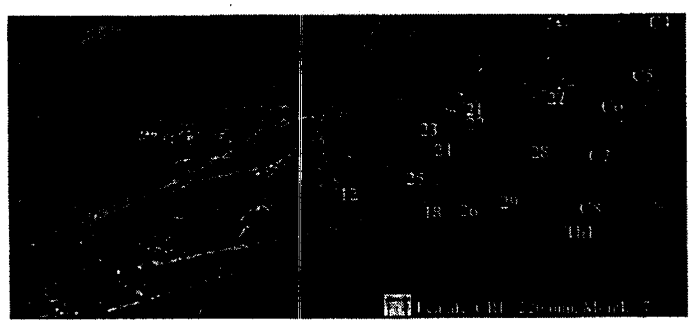


Fig. 13 Left body site: anastomosis between the musculocutaneous nerve (*8*) and radial nerve (*10*) (*red star*), connections between the anterior division of the middle trunk (ADMT; *23*) and median nerve roots (*green stars*)

of them presented more than one structural abnormality. Morphological variants of the following structures were detected: nerves roots, 35; trunks, 12; divisions, 74; cords, 40; distal branches, 35. Particular divisions of trunk variants were the most common anatomical anomalies, which occurred in every third plexus (33.63 %). Uysal et al. (2003) observed a similar anomaly prevalence. They examined 200 fetal brachial plexuses, and structural variants were observed in 107 cases (53.50 %). The authors found sexual dimorphism and asymmetry as anomalies occurred more often in females and on the right side.

In recent papers, morphogenesis of plexuses other than brachial was also assessed. Şişu et al. (2007) described the following steps for the ontogenegesis of the celiac ganglia. O'Rahilly et al. (1990) investigated the development of the thoracolumbar and sacrococcygeal regions of the human vertebral column on six embryos of 8 postovulary weeks (stage 23). The authors concluded that the dorsal rami of the thoracic nerves pass between the transverse processes and the tubercles of the ribs and then divide. The ventral rami of lumbar nerves 1 and 2 resemble the thoracic in their course, whereas those of nerves 3–5 are similar to the sacral. In at least some instances rudiments of coccygeal nerves 3–5 were detectable. Most papers describe plexuses obtained from adults. Matejcik (2003, 2005) carried out a complex evaluation of the brachial plexus in adults. In other available reports, the authors describe individual cases or selected plexus parts: Bhat and Girijavallabhan (2008), Gu (2007), Jahanshahi et al. (2003), Kocabiyik et al. (2005), Nayak et al. (2005), as well as Villamare et al. (2009). Matejcik (2005) examined 110 brachial plexuses coming from 55 adults corpses. Anomalies were observed in 83.6 % of plexuses. They appeared on the left side in 41.2 % and bilaterally in 33.6 % of the cases. A small number of examined plexuses may result in a high percentage of anomalies. A smaller number of anomalies is proved in the paper by Oliveira-Filho et al. (2009) who observed anatomical variations in 48 % of cases. In turn, Adebisi and Singh (2002) detected 72 % of brachial plexus structure anomalies in 90 adult Nigerians.

In our examined material, 185 brachial plexuses were formed by C_5, C_6, C_7, C_8, and Th_1 nerve root ventral branches. The C_4 nerve root occurred most often. In his paper, Lewis (1902) discussed the C_4 nerve root part in brachial plexus germ formation in the 4th week of the early embryonic period. Around the 5th week, the root separates and does not form the plexus any longer. These observations may account for the prevalence of C_4 nerve root ventral branches as persisting from the early embryonic period. Uysal et al. (2003) observed typical plexus root structure in 71.5 % of cases. In 25.5 % of plexuses, the C_4 nerve root appeared, and the Th_2 nerve root was present in 2.5 % of cases. In one case, both supernumerary roots were found. Kerr (1918) divided the examined plexuses into three groups: I, the group with the C_4 nerve root (62.85 %); II, the group without the C_4 nerve root but with the whole C_5 nerve root (29.79 %); III, the group in which the C_5 nerve root only partially forms the brachial plexus (7.42 %). In turn, the Th_2 nerve root was found in 30 % of cases.

Matejcik (2005) examined 100 plexuses coming from dead patients, and the C_4 accessory root was found in 24 cases. The Th_2 root was detected in only one plexus. The author revealed abnormalities similar to these described in the present article, such as the C_7–C_8 root connection. Uzun and Bilgic (1999) carried out autopsical examinations of 65 infant plexuses. The C_4 nerve root participated in the formation of 30.77 % of plexuses. Lee et al. (1992) analyzed the morphology of 152 brachial plexuses coming from 77 adult Koreans. In 21.7 % of plexuses, the C_4 nerve root appeared, and Th_2, C_4, and Th_2 nerves root prevalence was observed in single cases. In the autopsical material coming from 90 corpses of adults Nigerians, Adebisi and Singh (2002) observed C_4 nerve roots in 22.2 % of cases. These anomalies were regarded as important from the clinical point of view.

Trunk variants found in 12 cases were the most sporadic anomalies observed during our studies. Villamare et al. (2009) also observed the lack of the upper trunk during a routine autopsy in a 55-year-old woman. On the left side, the C_5 and C_6 nerve roots did not fuse into a common trunk, but they gave off anterior and posterior divisions that formed the lateral and posterior cords. Nayak et al. (2005) described an upper trunk variant that had been formed by C_5, C_6, and C_7 nerve roots. In turn, Matejcik (2003) found trunk anomalies in 25 plexuses: 11 on the left side and 7 bilaterally.

Division variations were the most common anomalies in our surveys. Similar anomalies of the ADMT were observed by Uysal et al. (2003). In their studies, division anomalies occurred most often, especially the ADMT (23 % of cases). In 2.5 % of cases, the ADMT formed the third median nerve root. Besides, in 1.5 % of their observations, the authors found an additional connection between the ADMT and ulnar nerve.

Morphological variations of cords were found in 40 cases—18.18 % of the examined plexuses. No anomalies of the medial cord were found; however, they have been described in the literature (Matejcik 2003; Uzun and Bilgic 1999). Uysal et al. (2003) discussed the lateral cord formed by the anterior division of the upper trunk in 2.5 % of cases as well as an accessory connection between the lateral cord and medial root of the median nerve in 5 % of observations. In the material composed of 175 plexuses, Kerr (1918) observed no lateral cord in 7 cases and no typical posterior cord (formed by radial nerve only) in 36 plexuses. In 99.46 % of cases, the medial cord was formed mainly by anterior division of the lower trunk, and in only two cases was the medial cord cocreated by the posterior division of the middle trunk. Matejcik (2003) found 22 cord anomalies that concerned the lateral cord most often. Also Uzun and Bilgic (1999) described cord variants, and in 4 of them (3.07 %), the lateral cord was formed by anterior division of the upper trunk only. Bhat and Girijavallabhan (2008) discussed a case where after the posterior cord formation, the cord subdivided into two roots that pressed and closed the subclavicular artery. Subsequently, the roots united and formed the radial nerve. Satyanarayana et al. (2009) also pointed out the clinical role of nervous and vascular conflicts.

Distal ramification anomalies were observed quite often—in 15.9 % of plexuses. Kerr (1918) described similar anastomoses between nerves. In their surveys carried on newborn babies, Uzun and Bilgic (1999) described the prevalence of a median nerve triple root in 4 out of 130 examined plexuses. Matejcik (2003) observed terminal nerve anomalies in 60 cases. The axillary nerve went off the posterior division of the upper trunk in three cases, and the radial nerve united with the ulnar one in two plexuses. The following anastomoses were described: musculocutaneous and median nerve anastomosis (6 observations); ulnar and antebrachium medial cutaneous nerve anastomosis (1 plexus); lateral, posterior, or medial cord and median nerve anastomosis (several cases). Median nerve roots combined in the brachium inferior part in 9 out of 110 examined plexuses (8.18 %). Pandey and Shukla (2007), Aktan et al. (2001), Kaus and Wójtowicz (1995), Uzun and Seelig (2001), Singhal et al. (2007), and Badawoud (2003) presented the prevalence of a connective branch between the median and musculocutaneous nerves. The radial nerve was found to go off the posterior cord, which was formed by posterior divisions of the lower and middle trunks. Ozguner et al. (2010) observed a connection between the ulnar and radial nerves during the routine autopsy of a 75-year-old male.

The fetal brachial plexus was characterized for anatomical structural variability. This variability seems to be independent from the plexus growing process. Series of recent papers show that the anatomical variants of the brachial plexus that developed in fetal life are observed in adults, e.g., Matejcik (2003, 2005), Villamare et al. (2009), and Oliveira-Filho et al. (2009).

In almost every third plexus, we detected a division anomaly. Structural variants of cords, nerve roots, or terminal nerves were observed very often. Anomalies occurred independently of sex or body side except for the cords, which were seen mainly on the left. Recognizing brachial plexus variants is important from the surgical and traumatological point of view.

Diversity of morphological variants could have some influence on the typology of the surrounding muscles.

In recent papers, cases of morphological variants of nerves together with surrounding muscles were discussed by Yogesh et al. (2010), Loukas and Aqueelah (2005), Tatar et al. (2004), Poornima and Satyaprasad (2006), and Loukas and Aqueelah (2005).

Four cases of correlation between different variant musculocutaneous and median nerves as well as the coracobrachialis muscle were described. Tatar et al. (2004) described the topography of innervation of the coracobrachialis muscle by a division from the lateral root of the median nerve. Poornima and Satyaprasad (2006) reported the case of a unique combination of muscle and nerve variants: the absence of the musculocutaneous nerve bilaterally and the presence of a third head of the biceps brachii in the left arm. Also divisions of the median nerve in a single cadaver innervated all the flexors in both arms.

These examples show how clinically important knowledge about nerve variants and the surrounding muscles can be. Morphological variability should be the reason for presurgical imaging of the brachial plexus.

Conflict Interest None.

References

Adebisi A, Singh SP (2002) Anomalous patterns of formation and distribution of the brachial plexus in Nigerians and the implication for brachial plexus block. Niger J Surg Res 4:103–106

Aktan ZA, Ozturg L, Bilge O, Ozer MA, Pinar YA (2001) A cadaveric study of the anatomic variations of the brachial plexus nerves in the axillary region and arm. Turk J Med Sci 31:147–150

Badawoud M (2003) A study of anatomical variations of median nerve formation. Bahrain Med Bull 25(4):1–9

Bhat KMR, Girijavallabhan V (2008) Variation in the branching pattern of posterior cord of brachial plexus. Neuroanatomy 7:10–11

Carlson BM (1999) Human embryology and developmental biology. Mosby, St Louis, p 450

Castellana C, Kosa F (1999) Morphology of the cervical vertebrae in fetal-neonatal human skeleton. J Anat 194:147–152

Gu Y (2007) Contralateral C7 root transfer over the last 20 year in China. Chin Med J 120(3):113–1126

Jahanshahi M, Moharreri AR, Golalipour MJ (2003) A variation of brachial plexus: absence of musculocutaneous nerve. MJIH 6(1):87–88

Kaus M, Wójtowicz Z (1995) Communicating branch between the musculocutaneous and median nerves in human. Folia Morphol 54(4):273–277

Kerr AT (1918) The brachial plexus of nerves in man, the variation and branches. Am J Anat 23:285–395

Kocabiyik N, Palcin B, Yazar F, Ozan H (2005) An accessory branch of musculocutaneous nerve joining median nerve. Neuroanatomy 4:13–15

Lee HY, Chung IH, Sir WS, Kang HS, Lee HS, Ko JS, Lee MS, Park AS (1992) Variations of ventral rami of the brachial plexus. Korean Med Sci 7(1):19–24

Lewis WH (1902) The development of the arm in man. Am J Anat 1(2):145–183

Loukas M, Aqueelah H (2005) Musculocutaneus and median nerve connections within, proximal and distal part to the coracobrachialis muscle. Folia Morphol 64(2):101–108

Matejcik V (2003) Aberrant formation and clinical picture of brachial plexus from the point of view of a neurosurgeon. Bratisl Lek Listy 104(10):291–299

Matejcik V (2005) Variations of nerve root of the brachial plexus. Bratisl Lek Listy 106(1):34–38

Nayak S, Somayaji N, Vollala VR, Reghunthan D, Rodrigues V (2005) A rare variation in the formation of upper trunk of the brachial plexus—a case report. Neuroanatomy 4:37–38

Oliveira-Filho J, Araujo VF, Queiroz RS, Nunes LS, Masuko TS (2009) Brachial plexus variations: an anatomic study. R Ci Med Biol Salvador 8(2):142–145

O'Rahilly R, Müller F, Meyer DB (1990a) The human vertebral column at the end of the embryonic period proper. 3. The thoracicolumbar region. J Anat 168:81–93

O'Rahilly R, Müller F, Meyer DB (1990b) The human vertebral column at the end of the embryonic period proper. 4. The sacrococcygeal region. J Anat 168:95–111

Ozguner G, Desdicioglu K, Albay S (2010) Connection between radial and ulnar nerves at high humeral level. Int J Anat Var 3:49–50

Pandey SK, Shukla VK (2007) Anatomical variations of the cords of brachial plexus and median nerve. Clin Anat 20:150–156

Poornima GC, Satyaprasad V (2006) Variant innervation of flexors of the arm associated with additional head of biceps brachii. Neuroanatomy 5:24–26

Rodriguez-Niedenführ M, Burton GJ, Deu J, Sañudo JR (2001) Development of the arterial pattern in the upper limb of staged human embryos: normal development and anatomic variations. J Anat 199:407–417

Rodriguez-Niedenführ M, Vazquez T, Parkin I, Sañudo JR (2003) Arterial patterns of the human upper limbs: update of anatomical variations and embryological development. Eur J Anat suppl 1:21–28

Satyanarayana N, Vishwakarma N, Kumar GP, Guha R, Datta AK, Sunitha P (2009) Variation in relation of cords of brachial plexus and their branches with axillary and brachial arteries—a case report. Nepal Med Coll J 11(1):69–72

Shinohara H, Naora H, Hashimoto R, Hatta T, Tanaka O (1990) Development of the innervation pattern in the upper limb staged human embryos. Acta Anat 138:265–269

Singhal S, Rao VV, Ravindranath R (2007) Variations in brachial plexus and the relationship of median nerve with the axillary artery: a case report. J Brachial Plex Peripher Nerve Inj 2:21

Şişu AM, Petrescu CI, Cebzan CC, Niculescu MC, Niculescu V (2007) Study of celiac ganglia development. Rom J Morphol Embryol 48(1):55–58

Tatar I, Brohi R, Sen F, Tonak A, Celik H (2004) Innervation of the coracobrachialis muscle by a branch from the lateral root of the median nerve. Folia Morphol 63(4):503–506

Uysal II, Seker M, Karabulut AK, Büyükmumcu M, Ziylan T (2003) Brachial plexus variations in human fetuses. Neurosurgery 53(3):676–684

Uzmansel D, Kortoğlu Z, Kara A, Öztürk NC (2010) Frequency, anatomical properties and innervation of axillary arch and its relation to the brachial plexus in human fetuses. Surg Radiol Anat 32(9):859–863

Uzun A, Bilgic S (1999) Some variations in the formation of the brachial plexus in infants. Turk J Med Sci 29:573–577

Uzun A, Seelig LL Jr (2001) A variation in the formation of the median nerve: communication branch between the musculocutaneous and median nerves in man. Folia Morphol 60(2):99–101

Villamare J, Goodwin S, Hincke M, Jalali A (2009) A brachial plexus variation characterized by the absence of the superior trunk. Neuroanatomy 8:4–6

Yogesh AS, Joshi M, Chimurkar VK, Marathe RR (2010) Unilateral variant motor innervations of flexure muscles of arm. J Neurosci Rural Pract 1(1):51–53

EXHIBIT 9

SUPERIOR COURT OF WASHINGTON, LEWIS COUNTY

SCOTT HAMILTON, as guardian ad litem for Z.H.,	)	
Plaintiff,	)	
vs.	)	20-2-00543-21
LINDA AMONDSON-MULLER, Personal Representative of the ESTATE OF LAURA HAMILTON,	)	
Defendant.	)	

REMOTE DEPOSITION UPON ORAL EXAMINATION OF

ERIC KNOWLES

2:00 p.m.

APRIL 13, 2022

EDMONDS, WASHINGTON

(Via Zoom)

REPORTED BY: ELEANOR J. MITCHELL, RPR, CCR 3006

Q. (BY MS. MACHLER.) Okay. Well, are you going to testify that there is no loss of earning capacity?

A. I've just been asked to respond to Mr. Brandt's report, and Mr. Brandt has no loss of earnings capacity in his report and with economic damages.

Q. But he didn't examine the effect of a disability on earning capacity either, did he?

A. I don't recall. I'm not intimately knowledgeable of Mr. Brandt's report. But -- but again, I was asked to -- I was asked to review and respond to the plaintiff's economist's report.

Mr. Brandt does not present any loss of future earnings capacity, so as a result, I haven't -- I haven't looked at that.

Q. So let's go to the -- to your five things that you listed out. And the first one is regarding Zachary performing domestic services or lawn care services later to his expected life span.

So could you tell us what -- expand on that for us?

A. Certainly. So when I started in the consulting business, we did not have healthy life expectancy tables. The -- the company that provides those started that -- providing those estimates within over the last probably six or seven years.

And anyway, prior to this, having those tables, when projecting out future services in the way of, say, household services or chore services or, in this case, domestic services and garden -- garden services, there -- what we used to do is take a period of time at the conclusion of the life expectancy to take into consideration the frailty of elderly age with the idea that, when you're 74, 75, 76, 77, and into your 80s, whatever the live expectancy tables show for the individual, there is a period when one would expect he or she not to be able to provide those services in the home or out in the yard.

The -- the healthy life expectancy tables then came out with the sole purpose to attempt to measure that period of time when the -- when the individual no longer is fully functioning. And -- and so that -- those tables I've provided to you.

Mr. Brandt is very familiar with these tables and, I can tell you, has utilized them consistently in his cases. When defense counsel asked Mr. Brandt about the healthy life expectancy tables, he -- he did indicate that he had not used them for Zachary and didn't provide an explanation as to, you know, why he did not.

But -- but theoretically it's -- not accounting for those last few years prior to death is -- is inappropriate to consider that -- that Zachary would continue doing these types of services up through his date of death.

Q. Okay. So are you saying that he wouldn't need domestic services because he's automatically going to be in a nursing home? Or I'm not understanding what -- what the issue is here.

A. So the way that the domestic care and, more importantly, the gardening and lawn care services are presented, if -- if the incident never happened involving Zachary's delivery, if none of those things happened and Zachary has two healthy arms, what plaintiff experts are presenting is that he would have provided those services up through age 77 and his life expectancy.

What I've taken into consideration is that there -- there is a period of time at one's -- at the end of one's life that theoretically you're no longer going to be able to provide those services to -- to you and your family. So that hasn't been taken into consideration.

There's a period of time that, at the very end, that Mr. Brandt should consider zero services at the end of one's life. And that can be -- that can be a four, five-, six-, ten-, eleven-year period, depending upon the healthy life expectancy tables.

Q. So a four-, five-, or six-, eleven-year period where the person can't provide their own domestic services; is that what you saying?

A. Yes, it is.

Q. Okay. Well, isn't the point here in this case is that Zachary is never going to be able to provide himself with a lot of those services?

MS. MONIZ: Object to the form.

A. And I'm not saying that, Ms. Machler. What I'm saying to you is that the -- what is reasonable to present in the way of -- of domestic services and gardening services is for a lengthy period of time up until Zachary would -- would hit that elderly age.

So, say, if -- if it's, you know, a time period from, you know, twenty or thirty years through the age of, you know, 65 or 70, I think that's a reasonable presentation as far as a loss of services. I'm only commenting right now about that -- that latter portion of -- of one's life expectancy.

Q. (BY MS. MACHLER.) So if Zachary needs to have household services through his whole life, starting when he moves out of the house and his mom's not there to do it, and he needs those services, and we're talking about the cost of those services, how is it that, you know, in the few years at the end of his life we no longer need to account for the costs of those services that he's never been able to provide?

A. Because, Ms. Machler, if he had two healthy arms, he never would have provided those services at the end of his life. So if -- if he never would have been able to provide those services per the healthy life expectancy tables, then why -- why would he receive damages related to services he could never provide to himself at an elderly age?

Q. So -- all right. Well, let's just talk about an ordinary person who, you know, vacuums and now they reach the end of their healthy life expectancy, which is 68, are you saying that they'll never be able to vacuum again?

A. No. I'm not saying that. I'm saying on a more probable than not basis, we need to project out those services. And what the tables tell us is that, on average, people have a fully functioning healthy life expectancy and then they have a life expectancy. So you need to take into consideration that period of time.

I can't speculate as to each individual and their ability or inability to vacuum. I have to take into consideration that period of time that the tables note.

Q. Okay. And so what you're talking about, the healthy life expectancy, is that the person, when they reach the end of their healthy life expectancy, then they no longer can do certain things. Is that -- am I getting that correct?

A. Yes. That's correct.

Q. But it's not -- but that's not saying that when they reach the end of their healthy life expectancy that they don't -- that they no longer need those things.

A. Well, again -- and I've answered this already. The theory in presenting these types of services for -- for damage presentation is one would take a look at, if Zachary had two healthy arms, what period of time would Zachary have provided those services to he and his -- and his family versus -- versus what his needs are today.

And so my point is, again, Zachary, on a more probable than not basis, wouldn't have provided those services regardless of --

Q. Right. But I'm talking --

A. Regardless of whether he needs those services, Zachary would have been able to -- according to the tables, Zachary would not have been able to provide those.

Q. Okay. So I'm still -- I'm still not understanding how we can just cut off -- if we're talking about domestic services that Zachary needs and we're going to pay for them from his age of 21 to the end of his life expectancy, are you saying -- healthy life expectancy, are you saying that all of a sudden he's not going to need domestic services anymore?

A. No. I'm not saying that. I'm saying to you that at a certain point in the very far future, his healthy life expectancy will conclude. And we have to take into account that period of time where Zachary wouldn't have been able to provide those services anyway.

So -- so regardless of whether he needs them at some future age, the damage presentation should not include those services that we wouldn't have been able to provide anyway.

Q. Well -- okay. So what -- okay.

He wouldn't have been able to provide those anyway. So what do people do when they reach the end of their healthy life expectancy? Do they stop?

I mean, that -- so people have to do

something, don't they, when they reach the end of their healthy life expectancy? They either have to continue to do the best they can, or they have to go into a nursing home and every -- somebody does it for them. But people still need to have a bath, and they still need to have dinner, and they still need to have somebody do their laundry, don't they?

MS. MONIZ: Object to the form.

A. Correct. I can't speak to that age 'cause I'm not there yet. But I can say that people in their late 60s and 70s, when they're no longer healthy enough to provide those services in their home, they obviously would pay a third party to provide these services.

Q. (BY MS. MACHLER.) Right. And so why wouldn't we pay for Zachary to provide those services?

A. I believe I've answered -- I've answered this several times. It's the -- the concept that, if Zachary would already -- with two healthy arms, Zachary is going to be paying for those services at, say, age 68. Okay? Just using that an example.

So if Zachary with two healthy arms is paying for those services at age -- in his late 60s, then why should we pay -- why should -- why should we pay in the -- in the form of damages for those services if -- if Zachary would be already paying someone else to provide with two healthy arms.

Q. Okay. And in these healthy life expectancy tables, do they tell you what -- what at any -- individual at any one time is going to be unable to do at any given age?

A. No. The healthy life expectancy tables do not go into specifics or details about the services. They're just providing that period of time.

Q. Okay. But your second point was that you disagreed that Zachary needs domestic services or lawn care services starting at age 21. So could you expand on that for us?

A. This is a fairly logical, I think, self-explanatory issue. No one owns -- no one.

Very few people would own a home at age 21. And so when I looked at that and Ms. Johnson was -- in her life care plan was providing services to Zachary starting at age 21 related to, say, lawn care and gardening services, my reaction to that was, Well, no one's going to own their own home at age 21, or very few people do.

And so when I looked that up to get the actual, you know, median age, it's age 34. So roughly 13 years after Ms. Johnson has presented that -- that line item for service.

provide with two healthy arms.

Q. Okay. And in these healthy life expectancy tables, do they tell you what -- what at any -- individual at any one time is going to be unable to do at any given age?

A. No. The healthy life expectancy tables do not go into specifics or details about the services. They're just providing that period of time.

Q. Okay. But your second point was that you disagreed that Zachary needs domestic services or lawn care services starting at age 21. So could you expand on that for us?

A. This is a fairly logical, I think, self-explanatory issue. No one owns -- no one.

Very few people would own a home at age 21. And so when I looked at that and Ms. Johnson was -- in her life care plan was providing services to Zachary starting at age 21 related to, say, lawn care and gardening services, my reaction to that was, Well, no one's going to own their own home at age 21, or very few people do.

And so when I looked that up to get the actual, you know, median age, it's age 34. So roughly 13 years after Ms. Johnson has presented that -- that line item for service.

was alluding to the idea that he would have purchased a home.

Q. Okay. So that accounts for lawn care services. And so are you also going to testify that you disagree that Zachary would require domestic services starting at age 21?

A. No, I'm not, Ms. Machler. Because domestic services would -- would point more towards the things that Zachary could provide or perform inside his home. And I'm -- again, it's not my life care plan -- it's Ms. Johnson's -- but I would assume that's like laundry and cleaning and those things. And I assume those services could start -- would start at that young age.

Q. Okay. And then you have an issue about gardening and lawn care on a year-round basis.

A. Yeah. Mr. Brandt was asked about that in his deposition, and -- and he didn't take that into consideration. But I've done that in other cases I've worked on.

And there's obviously a seasonal aspect to lawn care and gardening. I -- I don't have anybody cut my yard between the months of November and March. So there is a seasonal aspect to those types of services and being offered and provided for.

And -- and as well as when I was mowing my own lawn, I didn't mow my lawn between March -- or November and March. So I think that not taking into consideration the seasonal aspect of those types of services is inappropriate.

Q. Okay. And then your fourth one was $35,000 for tuition, books, and supplies.

A. Yeah. I don't understand what this is for. You know, Ms. Johnson was asked about this in her deposition, and, you know, eventually she responded in her deposition on -- at page 79 that -- she said -- her response to the idea that -- that Zachary needs this $35,000 for tuition, books, and supplies would be provided to him to level -- to level out -- to level his impairment.

I'm using her words in that. I'm not sure what -- I think what she means is that -- she went on to explain that because Zachary doesn't have two healthy arms, he can't work during the course of his undergrad, and he can't then -- if he can't work, he can't pay for his -- his schooling.

And -- which didn't make any sense to me. And when I looked it up -- when I looked up the percentages of folks that actually, as -- as students, as young adults who pay for their own undergrad -- and I sent that to you -- it's 8 percent.