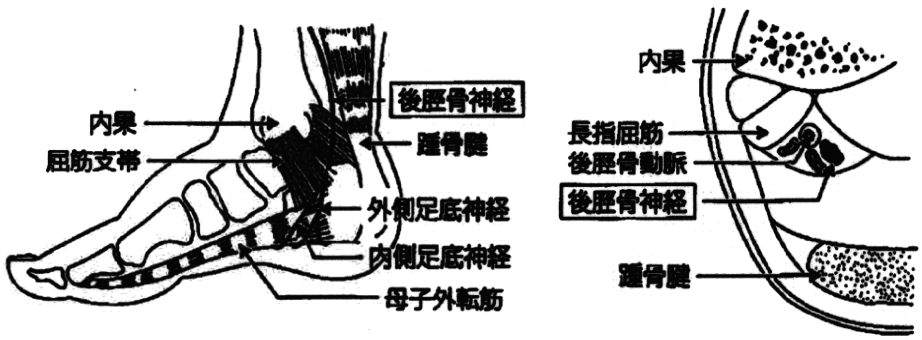


図5　後脛骨神経ブロックに必要な解剖

内果の1～3cm背側で，アキレス腱の少し前位を刺入点とし，針を少し前方に向けながら進めると筋膜を貫く感覚と放散痛を得ることができ，血液の逆流がないことを確認したのちに1%メピバカイン3mlを注入する．超音波ガイド下に行うと確実なブロックを行うことができる．患者の体位にはこだわらない．

（竹内　博．足関節，足底部，足背部の痛みに対するブロック．人間の医学 1998；33：508-11より改変引用）

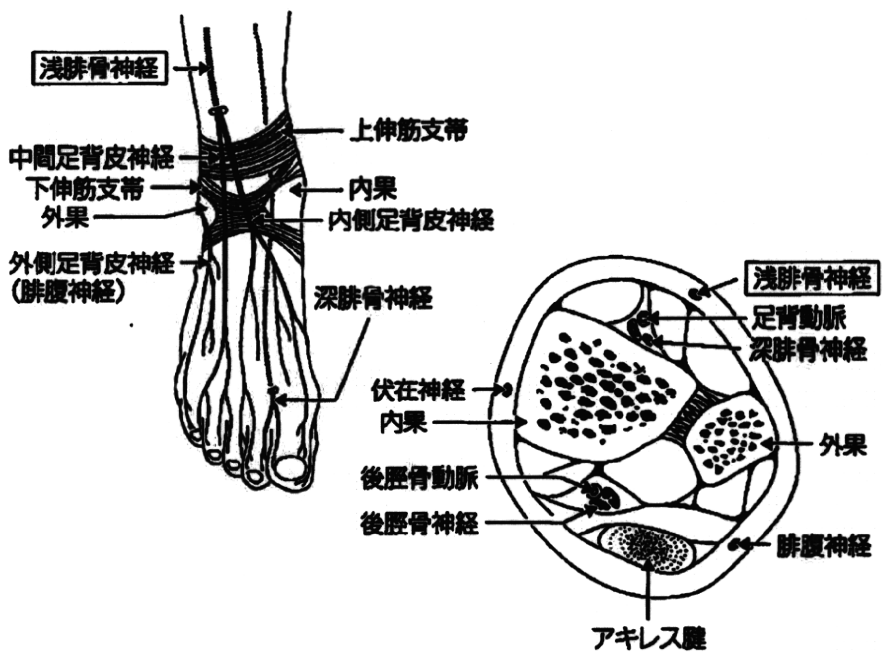


図6　浅腓骨神経ブロックに必要な解剖

患者を軽く膝関節を屈曲した状態の仰臥位とし，足関節から約10cm上方で腓骨前縁と腓骨外側縁の中間点を刺入点の目安とする．圧痛点があれば，皮膚に垂直に針を刺入，1cm程度進めると放散痛が得られる．血液の逆流がないことを確認したのちに1%メピバカイン3mlを注入する．超音波ガイド下に行うと確実なブロックを行うことができる．患者の体位にはこだわらない．

（竹内　博．足関節，足底部，足背部の痛みに対するブロック．人間の医学 1998；33：508-11より改変引用）

4. 予後，経過，次の手段

〈外科的治療〉

外科的治療が検討される進行例においても保存的治療は治療の基本で，保存的治療を継続しながら患者の全身状態を念頭に置いて外科的治療の適応を考える必要がある。外科的治療としては，関節鏡による遊離体摘出や滑膜切除，下位脛骨骨切り術，関節固定術，人工足関節置換術，骨・軟骨移植術などが行われている。外科的治療の選択にあたっては図8のフローチャート[4]を参考にするとよい。

B その他の疾患

1. リウマチ性足関節障害

関節リウマチでは，全身の関節の滑膜に炎症が出現し，滑膜増殖，関節包の肥大，靭帯の弛緩，関節軟骨の破壊，関節の強直による痛みと変形が生じる。足関節も足部とともにリウマチ病変の好発部位である（図9）[5]。両側の足首や足趾に痛みを感じ，腫れがあれば本症を疑う。20～50歳代の女性に多いといわれている。他の関節（特に手関節）の腫脹・変形，朝のこわばり，関節局所の熱感，皮下結節，血液検査など所見により他の疾患との鑑別は比較的容易である。

X線撮影は，関節の破壊程度，変形の評価，治療方針の決定に必要不可欠である。CTは骨性病変の評価，MRIは早期の診断に有用である。

治療は，関節リウマチの診断がつきしだい，抗リウマチ薬や抗炎症薬による全身管理を行うとともに，足関節障害に対しては変形性足関節症と同様に保存的治療を行う。特に関節リウマチの足関節障害は外反を呈することが多く，足底挿板などの装具療法が重要である。進行例に対しては関節鏡による滑膜切除術，関節固定術，人工足関節置換術などが行われている。

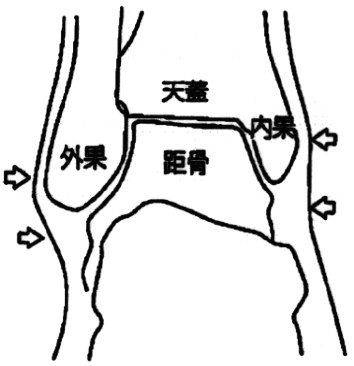


図7　足関節周囲のトリガーポイント

2. アキレス腱炎，アキレス腱周囲炎，滑液包炎

足関節周辺部の痛みの原因として比較的多くみられるのがアキレス腱炎，アキレス腱周囲炎，滑液包炎である。

アキレス腱炎は，普段運動をしていない人が運動を始めたときや，普段から運動を行っている人が運動量を急激に増やしたときにみられるアキレス腱そのものの炎症である。アキレス腱周囲炎は，アキレス腱周囲のパラテノン（アキレス腱は腱鞘をもたず，コラーゲンやエラスチンからなるパラテノンによって全長を覆われている）の炎症を指す。ともにアキレス腱周囲の腫脹・熱感・圧痛を認める。通常は，安静，運動量の調整，運動後のアイスマッサージなどにより軽快する。

アキレス腱滑液包炎は，アキレス腱の踵骨付着部の前方（踵骨後部滑液包）と後方（アキレス腱皮下滑液包）の炎症で（図10），靴による機械的刺激や踵骨の後上縁にできた骨棘などが原因となって発症することが多い。アキレス腱付着部に痛みを訴えることが多く，たいていは足底板を使用する，靴を変えるなどの生活上の工夫で軽快する。

これらの疾患で安静，足底板，靴の改良などでも痛みが持続する場合，アキレス腱周囲の圧痛点に局所注射することがある（図11）。

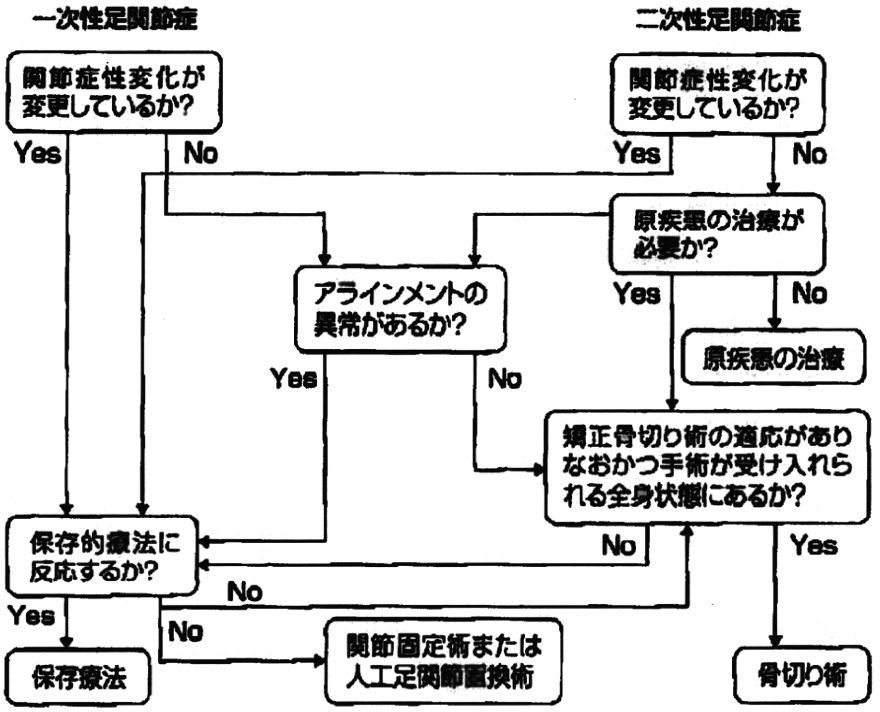


図8　外科的治療のフローチャート

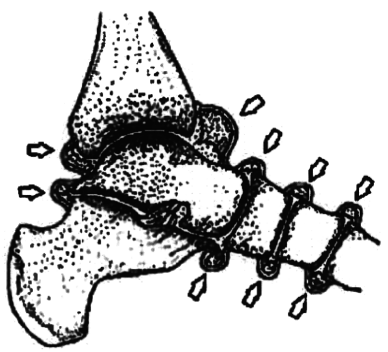


図9　足関節の関節リウマチの模式図

（寺山和雄，堀尾重治．足関節と踵部の疼痛と歩行障害．寺山和雄，堀尾重治編．図で説く整形外科疾患―外来診療のヒント．東京：医学書院；2005．p.155-60より改変引用）

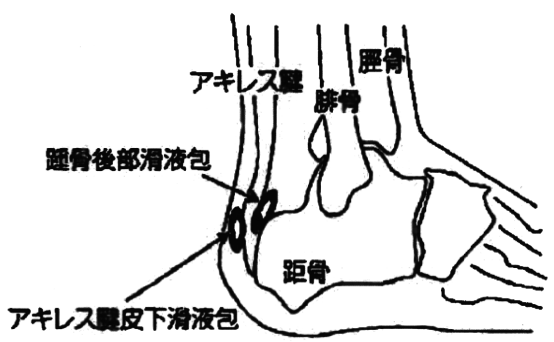


図10　アキレス腱付着部の滑液包

図11　アキレス腱周囲への局所注射

患者を腹臥位とし，術者が下腿を支持して足関節を背屈した状態に保持し，アキレス腱の遠位部の圧痛点を同定し，印を付ける．その部位のアキレス腱の両側から針を刺入し，アキレス腱の両脇で薬を注入する．薬液は片側1％メピバカイン1.5mlとデキサメタゾン0.5mlを使用する．アキレス腱は荷重のかかる腱であり，腱への直接穿刺は変性，断裂の原因となり，好ましくない．

3. 足根管症候群

脛骨内果，距骨，踵骨および屈筋支帯により構成される骨線維性トンネルである足根管（図12）[6]における脛骨神経の絞扼性障害で，内踝の下に限局性の圧痛を訴え，足底部から足趾にかけて放散痛を訴える。ガングリオン，足関節部の外傷や足根骨癒合症などが発症の原因となりうる。

ステロイド（デキサメタゾン1mg）と局所麻酔薬（1％メピバカイン0.5ml）の足根管内注入が有効なことが多く，難治例や再発を繰り返す症例では，原因検索を目的として手術が行われることもある。

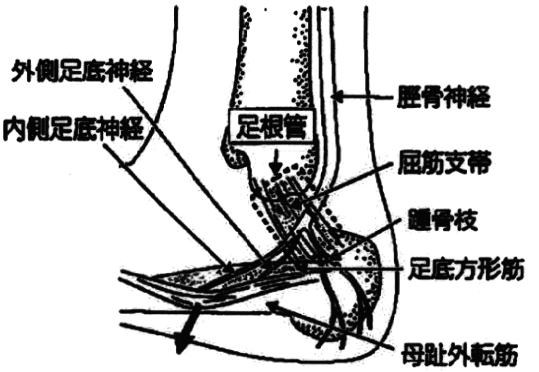


図12　足根管の解剖

脛骨神経は，脛骨内果，距骨，踵骨および屈筋支帯により構成される骨線維性トンネルである足根管を通る．
（佐藤勤也．足根管症候群．松崎昭夫編．新 図説整形外科講座 9.下腿・足．東京：メジカルビュー社；1994. p.150-1より改変引用）

4. 痛風

痛風発作は通常，足の第Ⅰ趾の付け根に起こることが多いが，足関節周辺にも起こることがある。夜間の急激な痛み，発赤と腫脹を伴い，暴飲暴食後に発症することが多い，血清尿酸値の上昇を伴うことなどにより診断は比較的容易である。

激痛は数日で軽快することが多く，痛みの軽減にはNSAIDsが使用される。高尿酸血症の治療を並行して行うことはいうまでもない。

[文　献]

(A 変形性足関節症)

1) 山本晴康．足関節の痛み．整形外科 2000；51：1075-82.
2) 塩見由紀代，山上裕章．肘関節，手関節，足関節などのブロック．大瀬戸清茂編．透視下神経ブロック．東京：医学書院；2009. p.179-82.
3) 竹内　博．足関節，足底部，足背部の痛みに対するブロック．人間の医学 1998；33：508-11.
4) 小島　朗．変形性足関節症．越智隆弘編．整形外科外来シリーズ11：足の外来．東京：メジカルビュー社；1999. p.176-80.

(B その他の疾患)

5) 寺山和雄，堀尾重治．足関節と踵部の疼痛と歩行障害．寺山和雄，堀尾重治編．図で説く整形外科疾患ー外来診療のヒント．東京：医学書院；2005. p.155-60.
6) 佐藤勤也．足根管症候群．松崎昭夫編．新 図説整形外科講座 9.下腿・足．東京：メジカルビュー社；1994. p.150-1.

〔山口重樹，北島敏光〕

1 神経ブロック療法

B 知覚神経ブロック

はじめに

癌性疼痛は，全身のあらゆる部位に生じる。ここでは，疼痛部位を2つに大別し，顔面・頭部と体幹・四肢に分けて詳述する。一般的には，まず局所麻酔薬による知覚神経ブロックを行い，これが有効な場合は神経破壊薬あるいは高周波熱凝固法を用いた神経ブロックを選択する。知覚神経ブロックによって疼痛が軽減されると，オピオイドなどの薬物の投与量が削減され，患者の生活の質（quality of life：QOL）が著しく改善されるので，適用があれば積極的に行うべきである。

顔面・頭部の癌性疼痛

顔面の癌性疼痛に対しては，三叉神経ブロックを行う。前額部には前頭神経ブロック，頬部には眼窩下神経ブロックあるいは上顎神経ブロック，下顎から耳介前面部には頤神経ブロック，耳介側頭神経ブロック，下顎神経ブロックを行う。顔面の広範囲な疼痛には，三叉神経節ブロックが勧められる。後頭部に疼痛を認める場合は，後頭神経ブロックを行う。

1 三叉神経ブロック

手技的には，末梢枝ブロックである前頭神経ブロック，眼窩下神経ブロック，頤神経ブロック，耳介側頭神経ブロックが簡単で，上顎神経ブロックと三叉神経節ブロックが難しく，かつ重篤な合併症にも注意が必要である。

a. 前頭神経ブロック[1)]

1）解　剖

前頭神経は三叉神経の第1枝，眼神経の枝で，眼窩上神経と滑車上神経とに分かれる。

眼窩上神経は外側枝と内側枝に分かれ，外側枝は眼窩上切痕，内側枝は前頭切痕を通って前頭部，前額部，上眼瞼，鼻根部，内眼角部の皮膚に分布する。

2) 手　技

患者をベッド上で仰臥位とし，眼窩の上壁で顔面の正中から約 2.5 cm 外側に眼窩上切痕のあることを確認する。術者は，消毒の際に薬液が眼内に入らないよう注意する。局所麻酔薬を充填した注射器のディスポーザブル針（23 あるいは 25G）を眼窩上切痕の上部で眉毛の上縁から皮膚面に対して直角に刺入する。針先が骨に当たり血液の逆流がないことを確認した後に，局所麻酔薬を眉毛に沿って左右に広がるように注入する。局所麻酔薬のみでブロックを行う場合は，0.75％ロピバカインまたは 0.5％ブピバカインなどの長時間作用性局所麻酔薬を約 2 ml 注入する。

神経破壊薬を用いる場合は，術者は滅菌手袋を着け，0.5％クロルヘキシジン・80％エタノール溶液を用いて消毒する。手技は前述のとおりに行い，2％リドカインまたは 2％メピバカイン 0.5 ml を注入し，前頭神経領域に無痛が得られ，かつ合併症がなければ局所麻酔薬注入 15 ～ 20 分後に 99.5％エタノールまたは 7 ～ 10％フェノール水溶液 0.5 ml を注入する。神経破壊薬の注入時に術者の左第 2 指を横にして眼窩上切痕のある眼窩の上壁をしっかりと押さえて薬液を左右に拡げ，抜針後も眼窩の上壁と針の刺入部を圧迫することによって，ブロック後の眼瞼腫脹や下垂を予防する。

3) 合併症

眼球損傷，眼瞼腫脹，眼瞼下垂，血腫，感染など。

b. 眼窩下神経ブロック[2)]

1) 解　剖

眼窩下神経は三叉神経の第 2 枝，上顎神経の枝で，眼窩下孔から出て，下眼瞼，鼻翼，上唇，鼻腔粘膜，上顎歯肉に分布する。

2) 手　技

患者をベッド上で仰臥位とし，眼窩の下壁約 1 cm 下で顔面の正中から約 2.5 cm 外側に眼窩下孔を確認する。術者は消毒の際に薬液が眼内に入らないように注意する。局所麻酔薬のみでブロックする場合は，注射器につけたディスポーザブル針（23 あるいは 25G）を眼窩下孔の下方で，やや外側から皮膚面に対して垂直に刺入する。針先を眼窩下孔に刺入することなく上顎骨に当て，血液の逆流がないことを確認して 0.75％ロピバカインまたは 0.5％ブピバカインなどの長時間作用性局所麻酔薬を約 2 ml 注入する。

神経破壊薬を用いる場合は，術者は滅菌手袋を着け，0.5％クロルヘキシジン・80％エタノール溶液を用いて消毒する。鼻翼の最外縁約 0.5 cm 耳側に局所麻酔薬を浸潤して膨疹を作り，ブロック針（22G，5 cm）の刺入点とする。眼窩下孔の位置を皮膚の上から左第 2 指でしっかりと触れておく。針先を上顎骨に当てるようにやや斜め上方に向かって刺入し，骨の上を滑らせるようにして眼窩下孔に向けて進め，針先を眼窩下管内

に刺入する。このとき，強い放散痛が得られる。針先が正しく眼窩下孔内に刺入されていると，術者がブロック針から手を離して，針を軽く上下動させても，針はしっかりと固定されている。2%リドカインまたは2%メピバカイン0.3～0.5 mlを注入し，無痛が得られ，かつ複視などの合併症がなければ，局所麻酔薬注入15～20分後に99.5%エタノールまたは7～10%フェノール水溶液を同量注入する。抜針後，ガーゼで眼窩下孔周囲を上からしっかりと圧迫し，ブロック後の頬部腫脹を予防する。

3) 合併症

顔面腫脹，血腫，複視，眼球損傷，視力障害，上顎洞穿刺，感染など。

c. 頤神経ブロック[3)]

1) 解　剖

三叉神経第3枝である下顎神経の末梢枝，下歯槽神経が頤孔から出て頤神経となり，下口唇および下顎の皮膚に分布する。

2) 手　技

患者をベッド上で仰臥位とし，顔面の正中から約2.5 cm外側に頤孔を確認する。局所麻酔薬のみでブロックする場合は，術者は消毒後に注射器につけたディスポーザブル針（23あるいは25G）を皮膚に対して垂直に刺入し，針先を骨に当てる。血液の逆流がないことを確認して頤孔周囲に0.75%ロピバカインまたは0.5%ブピバカインなどの長時間作用性局所麻酔薬を約2 ml注入する。

神経破壊薬を用いる場合は，術者は頤孔の位置を左第2指でしっかりと確認後に滅菌手袋を着け，0.5%クロルヘキシジン・80%エタノール溶液で消毒する。頤孔の耳側0.5 cm，上方0.5 cmから，注射器につけたディスポーザブル針（23あるいは25G）を皮膚に対して40～60°の角度で刺入し，針先を頤孔内に入れる。このとき，強い放散痛が得られる。また，術者が注射器から手を離しても，針は固定されている。2%リドカインまたは2%メピバカイン0.5 mlを注入し，無痛が得られ，かつ合併症がなければ，局所麻酔薬注入15～20分後に99.5%エタノールまたは7～10%フェノール水溶液を同量注入する。その際に，下顎部の腫脹を予防するためにガーゼで頤孔周囲を上からしっかりと圧迫する。

3) 合併症

下顎部腫脹，皮下出血，感染など。

d. 耳介側頭神経ブロック

1) 解　剖

耳介側頭神経は三叉神経第3枝，下顎神経の枝で，卵円孔から出た後に分枝して外側後方に進み，下顎骨関節突起の内側から浅側頭動脈の後方を並行して耳介前面と側頭部に分布する。

2) 手　技

患者をベッド上で仰臥位とし，術者の指で浅側頭動脈の拍動をしっかりと触知する。術者は消毒後に注射器につけたディスポーザブル針（23あるいは25G）を皮膚に対して垂直に刺入する。針先が皮下に刺入されたら，血液の逆流がないことを確認後に0.75％ロピバカインまたは0.5％ブピバカインなどの長時間作用性局所麻酔薬を約2 ml注入する。耳介側頭神経ブロックでは神経破壊薬を用いることはない。

3) 合併症

出血，感染，局所麻酔薬の血管内注入など。

e. 上顎神経ブロック

1) 解　剖

上顎神経は三叉神経の第2枝で，三叉神経節を起始として正円孔から頭蓋外に出る。上顎を支配し，頬部，鼻翼，上口唇，上顎粘膜・歯髄に分布する。

2) 手　技

患者をベッド上で仰臥位とし，患者の外眼角と外耳道を結ぶ線とベッドのなす角度が50～55°となるように顎を挙上させる。針の刺入点は頬骨弓下で耳珠軟骨基部から3 cmとする（図1）。術者は滅菌手袋を着け，0.5％クロルヘキシジン・80％エタノール溶液で消毒する。ブロック針（22G, 7 cm）の刺入点に局所麻酔薬を用いて膨疹を作る。X線透視下に，針先を皮膚に対して60～80°の角度で外眼角に向って刺入する。あらかじめ，針の刺入点から外眼角に向って線を描いておくとよい。また，ブロック針が眼窩内に刺入されないようにX線透視下に針先の方向を修正しながら，針をゆっくり進める。針が4.5～5.0 cm刺入され，鼻翼あるいは上口唇部に放散痛が得られたら，ブロッ

図1　上顎神経ブロック
針の刺入点は頬骨弓下で耳珠軟骨基部から3 cm。

ク針から造影剤 0.5 ml を注入すると，正円孔から眼窩下管入口部に向かう上顎神経に沿って造影剤が描出される。2％リドカインまたは 2％メピバカイン 0.5 ml を注入し，頬部，鼻翼，上口唇に無痛が得られ，かつ複視や脳神経障害などの合併症がなければ，局所麻酔薬注入 20 分後に 99.5％エタノールまたは 7～10％フェノール水溶液を同量注入する。

高周波熱凝固法を行う場合は，スライター針（22G，97 mm）を用いて同様の手技で行い，電気刺激によって放散痛を確認し，局所麻酔薬（2％メピバカイン 0.5 ml）によってこの反応が消失したら，凝固（90℃，90 秒）を行う。

3）合併症

出血，血腫，複視，顔面神経麻痺，感染など。

f. 下顎神経ブロック[4]

1）解　剖

下顎神経は三叉神経の第 3 枝で，三叉神経節を起始として卵円孔から頭蓋外に出て，下顎，舌，耳介前面，側頭部を支配する。

2）手　技

患者をベッド上で仰臥位とし，X 線透視あるいは撮影を行うために肩枕を入れて患者の頭部を懸垂頭位とする。外眼角と外耳道を結ぶ線に垂直となるように X 線の管球を合わせると卵円孔が描出できる。針の刺入点は頬骨弓下で耳珠軟骨基部から 2 cm とする（図 2）。術者は滅菌手袋を着け，0.5％クロルヘキシジン・80％エタノール溶液で消毒する。局所麻酔薬を用いて針の刺入点に膨疹を作り，ブロック針（22G，7 cm）を皮膚面に対して垂直に刺入する。針が 4.5 cm 程度刺入されると，下口唇または下顎に放散痛が得られる。次に，X 線透視あるいは写真によって針先が卵円孔直下であることを

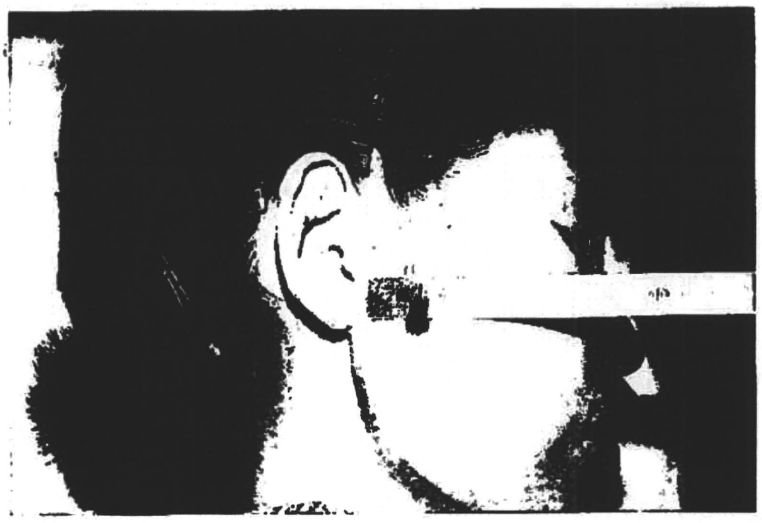


図 2　下顎神経ブロック
針の刺入点は頬骨弓下で耳珠軟骨基部から 2 cm。

確認する。2%リドカインまたは2%メピバカイン0.5 mlを注入し，下顎神経領域に無痛が得られ，さらに合併症がなければ，局所麻酔薬注入20分後に99.5%エタノールまたは7～10%フェノール水溶液を同量注入する。

高周波熱凝固法を行う場合は，スライター針（22G，97 mm）を用いて同様の手技で行い，電気刺激によって放散痛を確認し，局所麻酔薬（2%メピバカイン0.5 ml）によってこの反応が消失したら，凝固（90℃，90秒）を行う。

3）合併症

出血，血腫，咳嗽，嘔気・嘔吐，眼球振盪，顔面神経麻痺，感染など。

g. 三叉神経節ブロック

1）解　剖

三叉神経は混合性神経で，知覚性と運動性神経線維からなる。その知覚根と運動根は，ともに橋の両側で中小脳脚の前面から脳幹を出て三叉神経圧痕に行き，ここで知覚根が膨大して三叉神経節（半月神経節）となる。ここから3本の神経に分かれ，第1枝が眼神経，第2枝が上顎神経，第3枝が下顎神経となる。

2）手　技

患者をベッド上で仰臥位とし，X線透視あるいは撮影を行うために肩枕を入れて患者の頭部を懸垂頭位とする。外眼角と外耳道を結ぶ線に垂直となるようにX線の管球を合わせると卵円孔が描出できる。針の刺入点は口角から外側に引いた線と外眼角から垂直に下ろした線との交点とする。ブロック針（22G，10 cm）の刺入方向に必要な誘導線は，刺入点と瞳孔の中心を結ぶ線ならびに刺入点と耳介前方0.8 cmを結ぶ線とする（図3）。術者は滅菌手袋を着け，0.5%クロルヘキシジン・80%エタノール溶液で消毒する。局所麻酔薬を用いて針の刺入点に膨疹を作る。そこからX線透視下にブロック針を刺

図3　三叉神経節ブロック

針の刺入点は口角から外側に引いた線と外眼角から垂直に下ろした線との交点。

入するが，術者は針先を瞳孔に向って進め，また介助者は刺入点と耳介上部とを結んだ線にブロック針の方向が一致するように術者を誘導する。針先が下顎神経に達すると，下顎と舌に放散痛が得られる。そこからさらに針を進め，針先が卵円孔内に刺入されると，患者はブロック側の顔面全体に強い疼痛を訴える。次にブロック針の内針を抜いて脳脊髄液の逆流がないことを確認後に，2%リドカインまたはメピバカイン 0.2 ml を注入する。針先が正しい位置にあれば，局所麻酔薬注入直後からブロック側の顔面の痛覚は消失する。眼球振盪，複視，眩暈などの合併症がなければ，局所麻酔薬注入 20 分後に99.5%エタノールまたは7～10%フェノール水溶液を同量注入する。ブロック終了後，ベッド上で 1 時間安静臥床させる。

高周波熱凝固法を行う場合は，スライター針（22G，97 mm）を用いて同様の手技で行い，電気刺激によって放散痛を確認し，局所麻酔薬（2%メピバカイン 0.5 ml）によってこの反応が消失したら，凝固（90℃，90 秒）を行う。

3）合併症

角膜潰瘍，角膜炎，髄膜炎，脳神経障害，ghost pain など。

2 後頭神経ブロック

後頭部の疼痛に対しては，大後頭神経ブロックと小後頭神経ブロックがある。

a. 大後頭神経ブロック

1）解　剖

大後頭神経は第 2 頸神経の後枝からなり，後頭隆起の高さで正中から外側約 2.5 cm を後頭動脈とともに上行して，後頭部から頭頂部の知覚をつかさどる。

2）手　技

患者をベッド上で腹臥位とし，後頭隆起の正中から約 2.5 cm 外側を針の刺入点とする。術者は消毒後に注射器につけたディスポーザブル針（23 あるいは 25G）を皮膚に対して垂直に刺入する。針先を骨に当て，血液の逆流がないことを確認して 0.75%ロピバカインまたは 0.5%ブピバカインなどの長時間作用性局所麻酔薬を約 3 ml 注入する。なお，局所麻酔薬の注入部位がコブのように膨らまないようにするには，針先を上方に向けて刺入し，薬液が骨膜の上を這うように注入すればよい。大後頭神経ブロックでは神経破壊薬を用いることはない。

3）合併症

出血，感染，局所麻酔薬の血管内注入など。

b. 小後頭神経ブロック

1) 解　剖

小後頭神経は第2，3頸神経の前枝からなり，後頭隆起の高さで正中から外側約5 cmを上行して，耳後部から後頭部の知覚をつかさどる。

2) 手　技

患者をベッド上で腹臥位とし，後頭隆起の正中から約5 cm外側を針の刺入点とする。術者は消毒後に注射器につけたディスポーザブル針（23あるいは25G）を皮膚に対して垂直に刺入する。針先を骨に当て，血液の逆流がないことを確認して0.75％ロピバカインまたは0.5％ブピバカインなどの長時間作用性局所麻酔薬を約3 ml注入する。小後頭神経ブロックでは神経破壊薬を用いることはない。

3) 合併症

出血，感染，局所麻酔薬の血管内注入。

体幹・四肢の癌性疼痛

上肢の痛みに対しては腕神経叢ブロック，胸壁の疼痛に対しては肋間神経ブロック，体幹から下肢にかけた痛みに対しては持続硬膜外ブロックが有効である。体幹の限局した疼痛に対しては，恒久的な効果を得るためにくも膜下フェノールブロックを行うことがある。また，長期臥床による筋筋膜性疼痛に対して浅頸神経叢ブロック，深頸神経叢ブロック，肩甲上神経ブロック，脊髄神経後枝内側枝ブロック，トリガーポイント注射が有効なことが多い。

1 くも膜下フェノールブロック

体幹，会陰部，下肢の癌性疼痛に対して行う。

1) 解　剖

脊柱管の内壁は硬膜とくも膜が接着している。その内腔をくも膜下腔という。

2) 手　技

患者をベッド上で疼痛側を下にした側臥位とし，頭部に8から10 cm程度の枕を当てた後に患者に膝を抱え込ませ，臍を覗き込ませるように背中を丸くさせる。ブロックベッドを調整して疼痛部位を支配している脊髄神経の高さが最低位となるように患者の脊柱を“くの字”に彎曲させる。次に，患者の身体がベッドから転落しないように支持板で固定する。その後にベッドを患者の背側に45°回転させて半側臥位とし，疼痛部位

Copyeditor: Jamaica Polintan

ORIGINAL ARTICLE

Comparison of Bupivacaine, Ropivacaine, and Levobupivacaine in an Equal Dose and Concentration for Sympathetic Block in Dogs

Tomoki Hashimoto, MD, Tadaomi Iwasaki, MD, Yoshiyuki Takahashi, MD, Yoshiyuki Kimura, MD, Masaru Nagao, MD, Shigeki Yamaguchi, MD, Shinsuke Hamaguchi, MD, and Toshimitsu Kitajima, MD

Background and Objectives: The aim of this study was to compare the potency of bupivacaine, ropivacaine, and levobupivacaine in an equal dose and concentration for sympathetic block.

Methods: We measured mean arterial pressure, heart rate (HR), and right and left brachial artery blood flow (BABF) before and after cervicothoracic sympathetic block in 24 dogs. The experimental protocol was designed as follows: (1) left cervicothoracic sympathetic block with 1.0 mL of 0.25% bupivacaine (n = 8), (2) left cervicothoracic sympathetic block with 1.0 mL of 0.25% ropivacaine (n = 8), and (3) left cervicothoracic sympathetic block with 1.0 mL of 0.25% levobupivacaine (n = 8).

Results: Mean arterial pressure and heart rate did not change significantly throughout the study in either group. Left cervicothoracic sympathetic block with 0.25% bupivacaine increased left BABF significantly from 5 to 100 mins after the block (baseline, 100%; peak at 20 mins after the block, 218% ± 48%; $P < 0.01$). Left cervicothoracic sympathetic block with 0.25% ropivacaine increased left BABF significantly from 5 to 100 mins after the block (baseline, 100%; peak at 10 mins after the block, 254 ± 38%; $P < 0.01$). Left cervicothoracic sympathetic block with 0.25% levobupivacaine increased left BABF significantly from 5 to 80 mins after the block (baseline, 100%; peak at 20 mins after the block, 183% ± 38%; $P < 0.01$).

Conclusions: Ropivacaine may induce a greater increase in vasodilation than bupivacaine and levobupivacaine at the same dose and concentration for sympathetic block in dogs.

(*Reg Anesth Pain Med* 2010;35: 00–00)

Ropivacaine and levobupivacaine are long-acting aminoamide local anesthetic agents with a structure related to bupivacaine. Comparison of sensory and motor blocks with these 3 local anesthetics in equal concentrations has been examined in clinical studies. The onset of analgesia of levobupivacaine is similar or later compared with bupivacaine and ropivacaine.[1–3] The analgesic potency of bupivacaine is similar or greater compared with ropivacaine and levobupivacaine,[3,4] and ropivacaine may be the least potent among the 3 agents.[2] The degree of motor block of bupivacaine is greater compared with ropivacaine and levobupivacaine,[3–5] but comparison of ropivacaine and levobupivacaine for motor block is inconsistent.[1,6–8] However, to our knowledge, the potency of bupivacaine, ropivacaine, and levobupivacaine in an equal dose and concentration for sympathetic block have not been carefully studied. In the present study, we compared the vasodilative effects of bupivacaine, ropivacaine, and levobupivacaine in the same dose and concentration for sympathetic block in dogs.

From the Department of Anesthesiology, Dokkyo Medical University School of Medicine, Tochigi, Japan.
Accepted for publication January 29, 2010.
Address correspondence to: Toshimitsu Kitajima, MD, Department of Anesthesiology, Dokkyo Medical University School of Medicine, Mibu, Tochigi 321-0293, Japan (e-mail: kitajima@dokkyomed.ac.jp).

ISSN: 1098-7339
DOI: 10.1097/AAP.0b013e3181e6acf1

METHODS

This study was conducted according to the animal experimental guidelines of Dokkyo Medical University School of Medicine, which adhere to the National Institutes of Health's Animal Experimental Guidelines.

Twenty-four adult mongrel dogs of either sex (10–15 kg) were anesthetized with a 25-mg/kg intravenous injection of sodium pentobarbital, and the tracheas were intubated. Mechanical ventilation was adjusted to provide $PaCO_2$ between 35 and 40 mm Hg using a respirator (Harvard Apparatus, Chicago, Ill), and anesthesia was maintained with the intravenous administration of diazepam 0.05 mg/kg, pentazocine 0.5 mg/kg, and vecuronium 0.1 mg/kg, supplemented as required. The left femoral artery was cannulated with a polyethylene catheter (outer diameter, 2.75 mm) to measure mean arterial pressure (MAP) and to obtain blood samples for arterial blood gases. Electrocardiography was used throughout the experiment to monitor heart rate (HR). Bilateral brachial arteries in the forelegs were carefully dissected from the adjacent tissue, and a 2-mm ultrasonic flow probe (transonic system) was placed within each artery at the center of the proximal portion of the arteries. Right and left brachial artery blood flow (BABF) was measured using Transonic T206 (Transonic System, Inc, Ithaca, NY) as an ultrasonic timed flowmeter (mL/min). Physiologic saline solution was continuously infused intravenously at a rate of 3 mL/kg/hr from the left femoral vein during the study. The room temperature was kept constant at 25°C.

The left cervicothoracic sympathetic ganglion was then exposed by a left lateral thoracotomy at the second and third intercostal spaces. A 25-gauge winged needle was inserted under the fascia next to the ganglion with a suture for performing cervicothoracic sympathetic block, and the chest was closed.

After stabilization of hemodynamic parameters for 20 mins, the following baseline measurements were taken: MAP, HR, and left and right BABF. Using a computerized random number generator, the dogs were divided into 3 groups:

1. Left cervicothoracic sympathetic block with 1.0 mL of 0.25% bupivacaine (n = 8)
2. Left cervicothoracic sympathetic block with 1.0 mL of 0.25% ropivacaine (n = 8)
3. Left cervicothoracic sympathetic block with 1.0 mL of 0.25% levobupivacaine (n = 8)

Hemodynamic parameters were measured at 5 mins after the block, and thereafter every 10 mins for 120 mins after the

AQ1

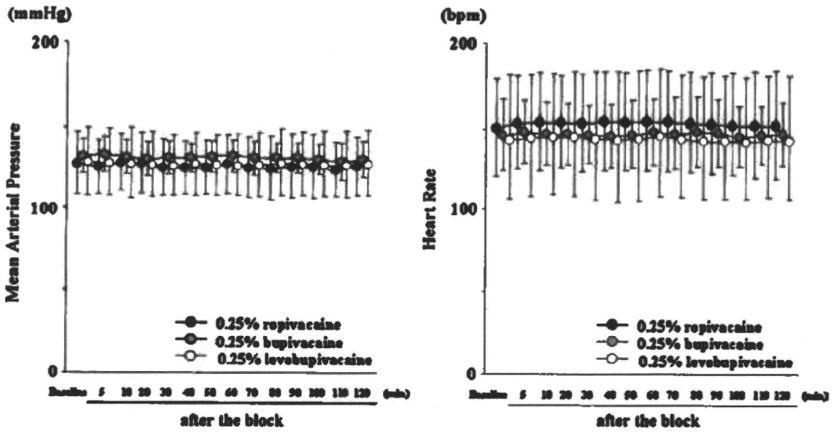


FIGURE 1. Changes in MAP and HR before and after cervicothoracic sympathetic block with 0.25% ropivacaine, 0.25% bupivacaine, or 0.25% levobupivacaine. Values are shown as mean ± SD.

block. All values of BABF were described as percentages of change from the baseline value (100%).

Blood gas analysis was performed before cervicothoracic sympathetic block (baseline) and at the end of the experiment.

Data are presented as mean ± SD. Statistical analyses within a group were performed by repeated-measures analysis of variance with Bonferroni correction of unpaired *t* test. Comparisons between the 2 groups were made by applying Mann-Whitney *U* test. The threshold for statistical significance was $P < 0.05$.

RESULTS

F1 F2 Mean arterial pressure and heart rate did not change significantly throughout the study in the 3 groups (Fig. 1). Figure 2 compares changes on left BABF after left cervicothoracic sympathetic block with bupivacaine, ropivacaine, or levobupivacaine. In cervicothoracic sympathetic block with 0.25% bupivacaine, left BABF increased significantly by 218% (baseline 100%; 20 mins after the block; $P < 0.01$) from 5 to 100 mins after the block compared with the baseline. In cervicothoracic sympathetic block with 0.25% ropivacaine, left BABF increased significantly by 254% (baseline 100%; 10 mins after the block; $P < 0.01$) from 5 to 100 mins after the block. In cervicothoracic sympathetic block with 0.25% levobupivacaine, left BABF increased significantly by 183% (baseline 100%; 20 mins after the block; $P < 0.01$) from 5 to 80 mins after the block. Left BABF in the left cervicothoracic sympathetic block with ropivacaine was the highest throughout the study in the 3 groups. Increases of BABF induced by sympathetic block with ropivacaine were significantly higher than those of bupivacaine at 30 and 40 mins after the block. Brachial artery blood flow induced by sympathetic block with ropivacaine also increased significantly compared with that of levobupivacaine from 5 to 120 mins after the block. As shown in Figure 3, right BABF in the 3 groups decreased after left cervicothoracic sympathetic block. There were no statistically significant differences between the groups. F3

$PaCO_2$ was maintained at 35 to 40 mm Hg, and PaO_2 was maintained at 80 to 98 mm Hg before the block and at the end of the experiment in the 3 groups.

DISCUSSION

Ropivacaine and levobupivacaine, long-acting local anesthetics, are useful alternatives to bupivacaine because of their lower cardiotoxicity and less toxicity of the central nervous system compared with bupivacaine.[9–12] However, little is known about the differential effects of ropivacaine and levobupivacaine when applied for sympathetic block. In the present study, we examined the duration and magnitude of the increase in vasodilation induced by cervicothoracic sympathetic block with ropivacaine or levobupivacaine and compared these results including bupivacaine. Cervicothoracic sympathetic block with 0.25% ropivacaine significantly increased both duration and magnitude of the vasodilative effect as compared with those of

AQ1

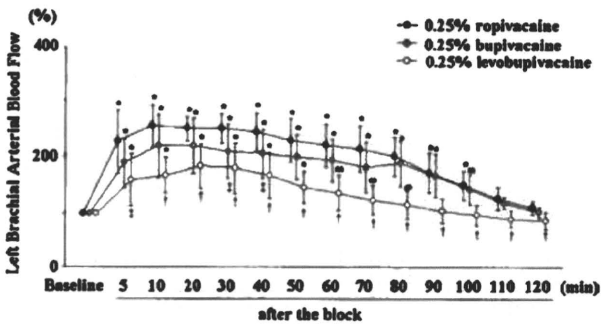
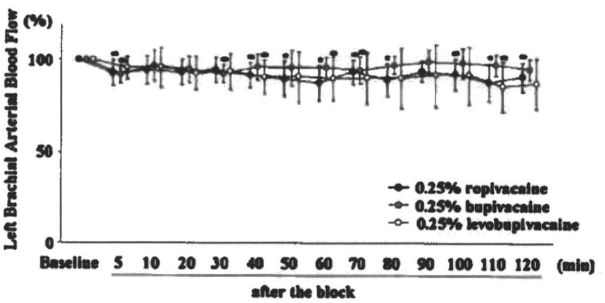


FIGURE 2. Changes in left BABF before and after cervicothoracic sympathetic block with 0.25% ropivacaine, 0.25% bupivacaine, or 0.25% levobupivacaine. *$P < 0.01$ versus baseline, **$P < 0.05$ versus baseline. †$P < 0.01$ versus ropivacaine, ‡$P < 0.05$ versus ropivacaine. Values are shown as mean ± SD.



AQ1

FIGURE 3. Changes in right BABF before and after cervicothoracic sympathetic block with 0.25% ropivacaine, 0.25% bupivacaine, or 0.25% levobupivacaine. *$P < 0.01$ versus baseline, **$P < 0.05$ versus baseline. Values are shown as mean ± SD.

0.25% levobupivacaine. Among the 3 local anesthetics, cervicothoracic sympathetic block with ropivacaine induced a greatest increase of BABF at an equal concentration. The least increase of BABF was observed in levobupivacaine. These results in dogs suggest that ropivacaine might be the preferred local anesthetic for sympathetic block in clinical practice. The onset time of sensory block and the duration of action of the 3 local anesthetics are different in clinical studies. The discrepancy between those reports may be explained by the difference in dose, concentration difference, and differences in experimental conditions, such as the method of regional anesthesia. González-Suárez et al[13] suggested that ropivacaine had a more selective action on nociceptive fibers (Aδ and C) than levobupivacaine because ropivacaine induced a faster onset of sensory block in axillary brachial plexus block. In the present study, the maximal increase of BABF induced by cervicothoracic sympathetic block with ropivacaine was faster than bupivacaine and levobupivacaine. Our experimental results may support their speculation because the postganglionic sympathetic fibers are unmyelinated C fibers.

In this study, no statistically significant differences were observed in MAP and HR after cervicothoracic sympathetic block. Because left cervicothoracic sympathetic block exerts less influence on hemodynamics compared with right-sided block,[14–17] left cervicothoracic sympathetic block was performed in this study. Schlack et al[17] demonstrated that left cervicothoracic sympathetic block impaired regional myocardial function, but it did not affect MAP and HR. Our results were consistent with their study.

In this study, cervicothoracic sympathetic block induced a decrease of contralateral BABF probably owing to compensatory vasoconstriction. We need to recognize this phenomenon in clinical practice.

Our model of experimental sympathetic block in dogs is different from that of clinical practice. In our study, the tip of the needle was placed adjacent to the canine cervicothoracic sympathetic ganglion, and 1.0 mL of local anesthetics was injected directly to the ganglion. After each experiment, 1.0 mL of methylene blue was injected through the needle to ascertain the spread of local anesthetics. We have demonstrated that 1.0 mL of a local anesthetic was sufficient for cervicothoracic sympathetic block in dogs when applied directly to the ganglion.[18–20] In a clinical setting, however, the tip of the needle rests on the anterior tubercle of the transverse process of the sixth or seventh cervical vertebra, and 5 to 10 mL of local anesthetics is injected around the ganglion. We believe that our results induced by canine cervicothoracic sympathetic block are more accurate compared with clinical cervicothoracic sympathetic block.

In conclusion, sympathetic block with ropivacaine may induce a greater increase in both duration and magnitude of peripheral arterial blood flow compared with bupivacaine or levobupivacaine in dogs. Levobupivacaine may be the least potent among the 3 local anesthetics at equal concentrations for sympathetic block.

REFERENCES

1. Casati A, Moizo E, Marchetti C, Vinciguerra F. A prospective, randomized, double-blind comparison of unilateral spinal anesthesia with hyperbaric bupivacaine, ropivacaine, or levobupivacaine for inguinal herniorrhaphy. *Anesth Analg*. 2004;99:1387–1392.
2. Ingelmo PM, Locatelli BG, Sonzogni V, et al. Caudal 0.2% ropivacaine is less effective during surgery than 0.2% levobupivacaine and 0.2% bupivacaine: a double-blind, randomized, controlled trial. *Paediatr Anaesth*. 2006;16:955–961.
3. Breschan C, Jost R, Krumpholz R, et al. A prospective study comparing the analgesic efficacy of levobupivacaine, ropivacaine and bupivacaine in pediatric patients undergoing caudal blockade. *Paediatr Anaesth*. 2005;15:301–306.
4. Camorcia M, Capogna G, Columb MO. Minimum local analgesic doses of ropivacaine, levobupivacaine, and bupivacaine for intrathecal labor analgesia. *Anesthesiology*. 2005;102:646–650.
5. Lacassie HJ, Habib AS, Lacassie HP, Columb MO. Motor blocking minimum local anesthetic concentrations of bupivacaine, levobupivacaine, and ropivacaine in labor. *Reg Anesth Pain Med*. 2007;32:323–329.
6. Beilin Y, Guinn NR, Bernstein HH, Zahn J, Hossain S, Bodian CA. Local anesthetics and mode of delivery: bupivacaine versus ropivacaine versus levobupivacaine. *Anesth Analg*. 2007;105:756–763.
7. Lim Y, Ocampo CE, Sia AT. A comparison of duration of analgesia of intrathecal 2.5 mg of bupivacaine, ropivacaine, and levobupivacaine in combined spinal epidural analgesia for patients in labor. *Anesth Analg*. 2004;98:235–239.
8. Ivani G, DeNegri P, Conio A, et al. Comparison of racemic bupivacaine, ropivacaine, and levo-bupivacaine for pediatric caudal anesthesia: effects on postoperative analgesia and motor block. *Reg Anesth Pain Med*. 2002;27:157–161.
9. Graf BM, Abraham I, Eberbach N, Kunst G, Stowe DF, Martin E. Differences in cardiotoxicity of bupivacaine and ropivacaine are the result of physicochemical and stereoselective properties. *Anesthesiology*. 2002;96:1427–1434.
10. Guinet P, Estebe J-P, Ratajczak-Enselme M, et al. Electrocardiographic and hemodynamic effects of intravenous infusion of bupivacaine, ropivacaine, levobupivacaine, and lidocaine in anesthetized ewes. *Reg Anesth Pain Med*. 2009;34:17–23.
11. Groban L. Central nervous system and cardiac effects from long-acting amide local anesthetic toxicity in the intact animal model. *Reg Anesth Pain Med*. 2003;28:3–11.
12. Casati A, Putzu M. Bupivacaine, levobupivacaine and ropivacaine: are they clinically different? *Best Pract Res Clin Anaesth*. 2005;19:247–268.
13. González-Suárez S, Pacheco M, Roigé J, Puig MM. Comparative study of ropivacaine 0.5% and levobupivacaine 0.33% in axillary brachial plexus block. *Reg Anesth Pain Med*. 2009;34:414–419.
14. Fujiki A, Masuda A, Inoue H. Effects of unilateral stellate ganglion block on the spectral characteristics of heart rate variability. *Jpn Circ J*. 1999;63:854–858.
15. Cinca J, Evangelista A, Montoya J, et al. Electrophysiologic effects of unilateral right and left stellate ganglion block on the human heart. *Am Heart J*. 1895;109:46–54.
16. Kashima T, Tanaka H, Minagoe S, Toda H. Electrocardiographic changes induced by the stellate ganglion block in normal subjects. *J Electrocardiol*. 1981;14:169–174.
17. Schlack W, Schäfer S, Thämer V. Left stellate ganglion block impairs left ventricular function. *Anesth Analg*. 1994;79:1082–1088.
18. Kitajima T, Okuda Y, Mishio M, Hamaguchi S, Yamaguchi S, Kimura Y. Acute cigarette smoking reduces vasodilative effect induced by sympathetic block in dogs. *Reg Anesth Pain Med*. 2001;26:41–45.
19. Takahashi Y, Yamaguchi S, Tezuka M, et al. Comparison of 0.2% ropivacaine, 0.125% bupivacaine, and 0.25% bupivacaine for duration and magnitude of action in peripheral arterial blood flow induced by sympathetic block in dogs. *Reg Anesth Pain Med*. 2004;29:441–445.
20. Iwasaki T, Takahashi Y, Kimura Y, Yamazaki H, Yamaguchi S, Kitajima T. Comparison of 0.25% levobupivacaine, 0.25% bupivacaine, and 0.125% bupivacaine for duration and magnitude of action in peripheral arterial blood flow induced by sympathetic block in dogs. *Reg Anesth Pain Med*. 2007;32:87–101.

Copyeditor: Josephine C. Gementera

ORIGINAL STUDY

Assessment of QT Interval and QT Dispersion During Electroconvulsive Therapy Using Computerized Measurements

Nobuko Tezuka, MD, Hirotoshi Egawa, MD, PhD,* Daigo Fukagawa, MD, PhD,* Shigeki Yamaguchi, MD, PhD,* Shinsuke Hamaguchi, MD, PhD,* Toshimitsu Kitajima, MD, PhD,* and Junichi Minami, MD, PhD†*

Background: Electroconvulsive therapy (ECT) used in the treatment of severe psychiatric disorders induces stimulation of the autonomic nervous system with initial parasympathetic outflow immediately followed by a sympathetic response. These responses induce an initial bradycardia, arrhythmias, and hypertension. QT dispersion (QTD), defined as maximal QT interval minus minimal QT interval on 12 leads of the surface electrocardiogram, reflects regional heterogeneity of ventricular repolarization. The effects of electrical stimulus due to ECT on QT interval and QTD are of considerable interest.

Objective: This study was designed to investigate the effects of electrical stimulation caused by ECT on RR interval, QT interval, the rate-corrected QT (QTc) interval, QTD, and the rate-corrected QTD (QTcD) under general anesthesia using computerized measurements.

Methods: Thirty psychiatric patients scheduled for ECT were studied under propofol anesthesia. A 12-lead electrocardiogram was monitored to measure parameters. Muscle paralysis was achieved by administering succinylcholine 1 mg/kg intravenously, and the efficacy of ECT was determined by the tourniquet technique.

Results: The RR interval and QT interval decreased significantly immediately after electrical stimulus, and returned to the baseline level 1 minute after electrical stimulus. In 25 out of 30 patients, the baseline value of QTc interval was higher than the normal limits, and the QTc interval decreased significantly for 2 minutes after electrical stimulus. In 27 out of 30 patients, the baseline values of QTD and QTcD were higher than the normal limits, and the QTD and QTcD increased significantly from immediately after electrical stimulus to 5 minutes after electrical stimulus.

Conclusions: The QTc interval, QTD, and QTcD, which were associated with increased risks of ventricular arrhythmias, increased significantly before anesthetic induction in patients with major depression. Electrical stimulus during ECT induced further increases of the QTD and QTcD.

Key Words: electroconvulsive therapy, QT interval, QT dispersion, propofol

(*J ECT* 2009;00: 00–00)

Dispersion of the QT interval (QTD), which is defined as maximal QT interval minus minimal QT interval, on 12 leads of the surface electrocardiogram (ECG) reflects regional heterogeneity of ventricular repolarization.[1] Prolongation of the QTD is associated with increased risk of ventricular arrhythmias and cardiovascular mortality.[2–9] It is well known that the QTD is regulated by not only heart rate but also autonomic tone.[10–14]

Although the exact mechanism of electroconvulsive therapy (ECT) is not elucidated, ECT has been widely used in the treatment of severe psychiatric disorders such as depression and schizophrenia. In the early days of ECT, complications such as trauma and fracture had been common. The use of intravenous anesthetics and neuromuscular blockades reduced such complications. Even if anesthesia management is appropriate, however, electrical current during ECT stimulates the autonomic nervous system and provokes acute cardiovascular response with initial parasympathetic outflow immediately followed by a sympathetic response.[15] These responses may induce arrhythmias or cardiac events. However, to our knowledge, QT interval and QTD during ECT have not been carefully measured using a computer. Computerized measurements enhance the accuracy and reproducibility compared with manual measurements. The purpose of this study was to investigate QT interval and QTD during ECT under propofol anesthesia using a computer.

METHODS

Thirty patients with major depression (22 women, 8 men; age, 40–64 years), American Society of Anesthesiologists 1 or 2, who were scheduled to undergo elective ECT under propofol anesthesia, were studied after approval of the hospital ethics committee had been obtained, and the patients or their family had given informed consent. All patients with cardiovascular or respiratory diseases were excluded from the study. To avoid enrollment of patients with cardiac diseases, all patients received echocardiography and ECG before the study. All patients received antidepressants, benzodiazepines, and/or major tranquilizers (Table 1). They received the usual long-term medication in the morning before ECT session. T1

No patients received premedication before entry to the operating room. After arriving at the operating room, standard 12-lead ECGs (FDX-4521L; Fukuda Denshi Co Ltd, Tokyo, Japan), indirect arterial blood pressure, pulse oximetry (Satlite; Datex, Finland), and capnography (Capnomac; Datex) were monitored. A tourniquet was applied above the right ankle. Anesthesia was induced with intravenous injection of propofol 1 mg/kg by a trained anesthesiologist. After loss of consciousness, ventilation was controlled using a face mask with 100% oxygen, and the end-tidal carbon dioxide partial pressure measured at nostril was maintained between 35 and 40 mm Hg. The tourniquet was simultaneously inflated to 300 mm Hg to isolate the circulation to the foot and permit accurate motor seizure assessment. Then, succinylcholine 1 mg/kg was administered intravenously. Immediately after fasciculation caused by succinylcholine injection disappeared from the left leg, the electrical stimulus was delivered via bitemporal electrodes by a single psychiatrist using an ECT stimulator (Thymatron System; AQ1

From the *Department of Anesthesiology, and †Department of Hypertension and Cardiorenal Medicine, Dokkyo Medical University School of Medicine, Mibu, Tochigi, Japan.
Received for publication February 03, 2009; accepted April 01, 2009.
Reprints: Toshimitsu Kitajima, MD, Department of Anesthesiology, Dokkyo Medical University School of Medicine, Mibu, Tochigi 321-0293, Japan (e-mail: kitajima@dokkyomed.ac.jp).

TABLE 1. List of All Medications

Antidepressants	
Clomipramine	n = 6
Amitriptyline	n = 2
Nortriptyline	n = 1
Amoxapine	n = 1
Mianserin	n = 9
Setiptiline	n = 1
Milnacipran	n = 11
Fluvoxamine	n = 3
Paroxetine	n = 5
Trazodone	n = 3
Lithium	n = 4
Benzodiazepines	
Etizolam	n = 2
Lorazepan	n = 3
Alprazolam	n = 6
Cloxazolam	n = 3
Triazolam	n = 2
Lormetazepan	n = 1
Brotizolam	n = 7
Flunitrazepan	n = 18
Estazolam	n = 4
Nitrazepan	n = 5
Quazepan	n = 1
Major Tranquilizers	
Aripiprazole	n = 2
Risperidone	n = 2
Quetiapine	n = 6
Chlorpromazine	n = 5
Levomepromazine	n = 2
Perphenazine	n = 1

AQ2 Somatics). The magnitude of the energy setting for ECT stimulus was predetermined by age. The efficacy of ECT was determined by the tourniquet technique. That was by observation of convulsive movements of the distal leg, around which an inflated tourniquet was set to block the distribution of succinylcholine. Electroencephalogram (EEG) seizure was also measured by an EEG monitor set in the electrical stimulator. The criteria for adequacy of electrical stimulus were more than 15 seconds of EEG seizures.

From each ECG, consecutive beat-to-beat data were digitally recorded at a sample rate of 2 milliseconds and stored on a 3.5-in. floppy disk. QT intervals were determined by the use of newly developed software (QTD-1; Fukuda Denshi Co. Ltd.) that detected the onset of the Q wave and the end of the T wave. The software used the differential threshold technique described elsewhere in detail.[16,17] In brief, this technique determines the onset of the Q wave as the intersection of a threshold level with the differential of the Q wave and the end of the T wave as the intersection of a threshold level with the differential of the T wave, respectively. QT intervals were measured in all 12 leads and corrected for heart rate (QTc) with Bazett's formula.[18] QTD was calculated as the difference between maximal and minimal QT intervals. The corrected QT dispersion (QTcD) was defined as the difference between the maximum and minimum average QTc interval in the 12-lead ECG. The average value of data derived 3 successive beats for each lead was used for analysis. A cardiologist performed all measurements and analysis. Leads in which the end of the T wave could not be clearly discerned were excluded from the study.

Measurements of the RR interval, QT interval, QTc interval, QTD, and QTcD were performed before anesthetic induction (baseline), immediately after anesthetic induction, immediately after electrical stimulus, and every 1 minute for 10 minutes after electrical stimulus.

Data are presented as mean ± SD. Differences were analyzed by 2-way repeated-measures analysis of variance. Scheffe's tests were used as determined by the analysis of variance results. The threshold for statistical significance was P less than 0.05.

RESULTS

All data were available in this study.

The RR interval did not change significantly after anesthetic induction. However, it shortened significantly immediately after electrical stimulus, and recovered the baseline value 1 minute after electrical stimulus (Fig. 1). F1

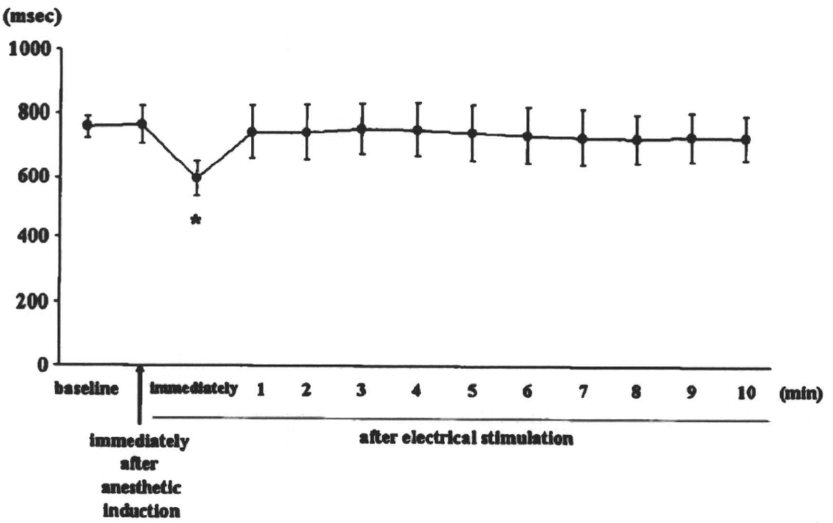


FIGURE 1. Changes in the RR interval. All values are expressed as mean ± SD. *P < 0.01 versus baseline.

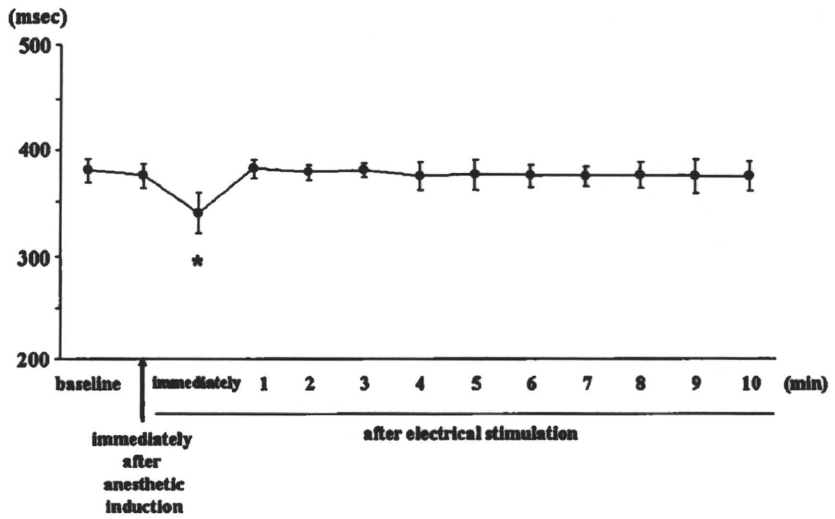


FIGURE 2. Changes in the QT interval. All values are expressed as mean ± SD. *$P < 0.01$ versus baseline.

A significant decrease of the QT interval occurred immediately after electrical stimulus, and recovered the baseline F2 value 1 minute after electrical stimulus (Fig. 2). In 25 out of 30 patients, the baseline value of QTc interval was higher than the normal limits (320–440 milliseconds). Significant decreases of the QTc interval were observed from immediately after electrical F3 stimulus to 2 minutes after electrical stimulus (Fig. 3).

In 27 out of 30 patients, the baseline values of QTD and QTcD were higher than the normal limits (20–50 milliseconds) F4 F5 in Figures 4 and 5. The QTD increased significantly from immediately after electrical stimulus to 5 minutes after electrical stimulus (baseline, 65.0 ± 8.5 milliseconds; peak at immediately after electrical stimulus, 95.9 ± 3.7 milliseconds; $P < 0.01$). The QTcD also increased significantly from immediately electrical stimulus to 5 minutes after the electrical stimulus (baseline, 73.8 ± 8.8 milliseconds; peak at immediately after the electrical stimulus, 125.2 ± 7.2 milliseconds; $P < 0.01$).

We observed temporary ventricular premature complexes (VPC) in 2 patients and sinus tachycardia in 24 patients after electrical stimulus.

DISCUSSION

The present study focused on computerized measurements of RR interval, QT interval, QTc interval, QTD, and QTcD during ECT under propofol anesthesia. QT interval is the time interval from the first recognizable part of QRS complex to the final recognizable part of the T wave. The QT interval is easy to measure because QT deflections are usually sharp. However, the terminal part of the T wave is often a rather gentle slope, and the precise position of the final part of T wave may be difficult or even impossible to determine.[19] Therefore, the accuracy and reproducibility of the QT interval and QTD in manual measurements have been limited. Use of computerized detection of T wave offset enhances more accuracy and reproducibility.

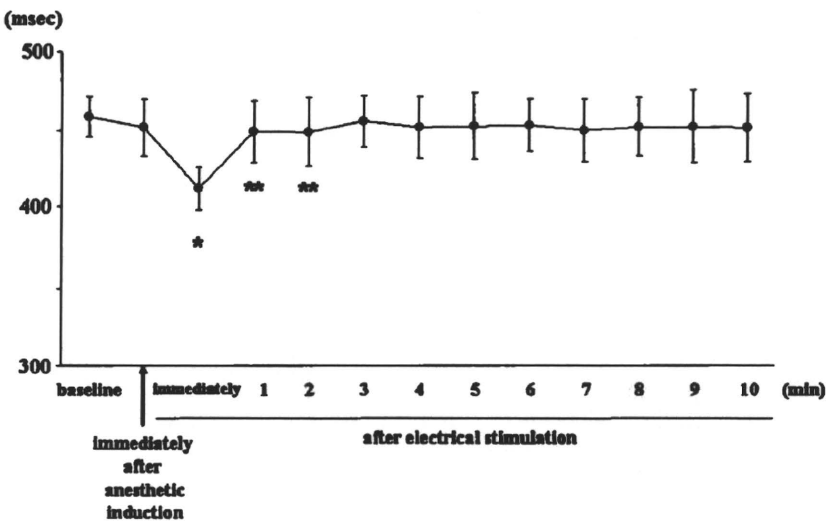


FIGURE 3. Changes in the QTc interval. All values are expressed as mean ± SD.

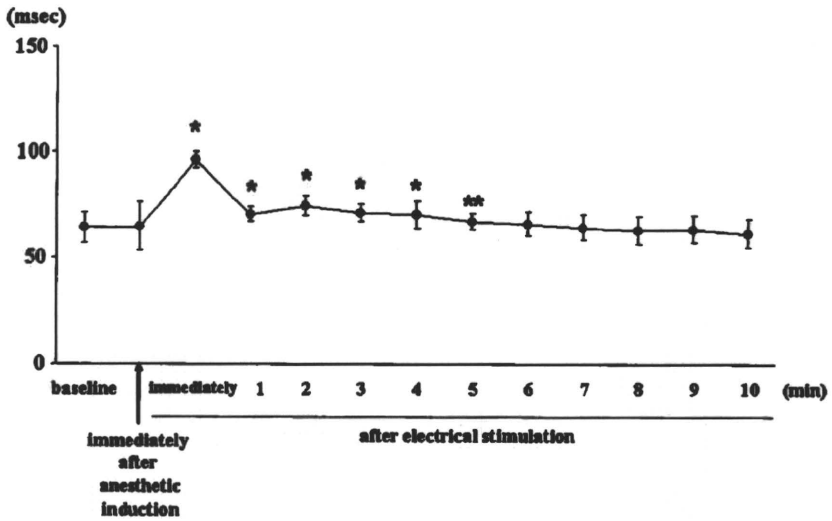


FIGURE 4. Changes in the QTD. All values are expressed as mean ± SD. *$P < 0.01$ versus baseline, **$P < 0.05$ vs baseline.

This is the first study to examine how ECT affects the ECG findings using computerized measurements. In the present study, the QT interval and QTc interval decreased significantly immediately after electrical stimulus. Because temporary predominance of parasympathetic nerve activity occurs during electrical stimulus, withdrawal of parasympathetic nerve activity may shorten the QT interval and QTc interval. This finding appears to be in agreement with earlier observation that withdrawal of parasympathetic nerve activity shortens the QT interval.[20] We also found that electrical stimulus during ECT caused increases of the QTD and QTcD in patients with major depression. Because the QTD and the QTcD have been shown to predispose to ventricular arrhythmia, our finding of changes in QTD and QTcD during ECT may explain the occasional emergence of ventricular arrhythmia during ECT. Temporary ventricular premature complexes or sinus tachycardia occurred after electrical stimulus in 26 of the 30 patients.

Interestingly, the baseline values of the QTc interval in 83% of patients in this study were already higher than the upper normal limit of 440 milliseconds. Tricyclic antidepressants are known to induce prolongation of the QT interval.[21] In the previous studies, use of tricyclic antidepressants, thioridazine, droperidol, or butyrophenone, is a robust predictor of QTc prolongation in a dose-dependent manner.[22,23] The baseline values of the QTD and QTcD in 90% of patients were also higher than the upper normal limits of 50 milliseconds. Rasmussen et al[24] reported that QTcD as measured on the baseline ECG positively correlated with number of arrhythmias during ECT. Although electrical stimulus during ECT caused further increases of the QTD and QTcD, these values returned to the baseline value 6 minutes after electrical stimulus in this study.

Selection and determination of dosage of anesthetic agents may be crucial in ECT management. Anesthetic requirements

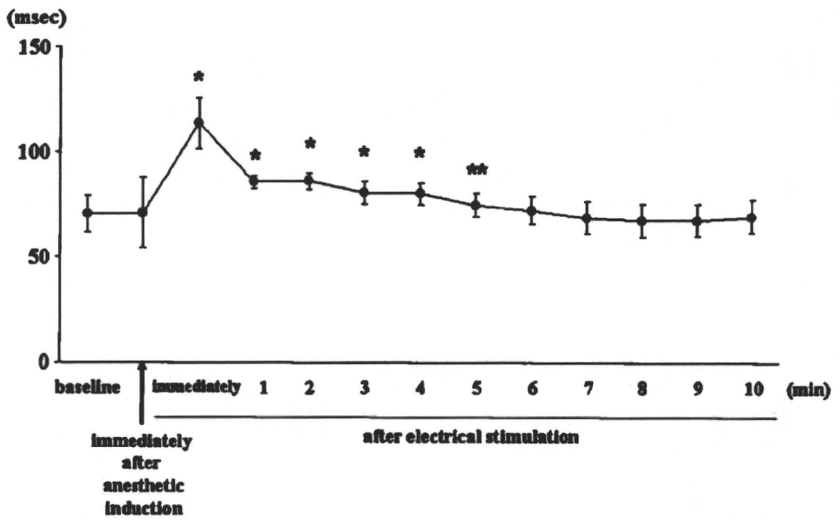


FIGURE 5. Changes in the QTcD. All values are expressed as mean ± SD. *$P < 0.01$ versus baseline, **$P < 0.05$ versus baseline.

for successful ECT are rapid induction, rapid recovery after the seizure, and minimization of any antagonistic effects on seizure activity by anesthetic agents.[15] We recommend intravenous ultra-short-acting anesthetics for ECT. Methohexital is recommended as the first choice anesthetic for ECT by the American Psychiatric Association.[15,25] Propofol is also used in ECT because systemic and cerebrovascular hemodynamic changes under propofol anesthesia are more stable than under barbiturate anesthesia. Kleinsasser et al[26] demonstrated that propofol using by 2.5 mg/kg shortened the QT interval but did not change the QTc interval. In the present study, however, the QT interval, QTc interval, QTD, and QTcD did not change after intravenous injection of propofol 1 mg/kg. The difference between the 2 studies may be because of the dosage of propofol. Volatile anesthetics such as sevofulurane are a suitable alternative treatment option to intravenous anesthetics.[27–31] Rasmussen et al[27] suggest that sevoflurane is useful for patients in whom intravenous access is problematic or in whom intravenous anesthetics cause severe on injection. As to neuromuscular blockade, succinylcholine 0.5 to 1.0 mg/kg has usually been used in ECT because of its short duration of action. It has to be deep enough to suppress abdominal muscle contraction to avoid aspiration of stomach contents, and to avoid trauma.[15]

In the present study, we excluded patients with cardiovascular diseases. Previous studies, however, have demonstrated that the QTD increases in patients with myocardial infarction, subarachnoid hemorrhage, or diabetes mellitus.[7,8,32–34] It is suggested that ECT may induce further increased risks of ventricular arrhythmias and cardiovascular events in such patients. Further examination is needed in such patients.

In conclusion, significant increases of the QTc interval, QTD, and QTcD, which are associated with increased risks of arrhythmias, were observed before anesthetic induction in patients with major depression. Electrical stimulus during ECT may induce further increases of the QTD and QTcD.

REFERENCES

1. Day CP, McComb JM, Campbell RWF. QT dispersion: an indication of arrhythmic risk in patients with long QT intervals. *Br Heart J.* 1990;63:342–344.
2. Priori SG, Napolitano C, Diehl L, et al. Dispersion of the QT interval: a marker of therapeutic efficacy on the idiopathic long QT interval. *Circulation.* 1994;89:1681–1689.
3. Lee KW, Okin PM, Kligfield P, et al. Precordial QT dispersion and inducible ventricular tachycardia. *Am Heart J.* 1997;134: 1005–1013.
4. de Bruyne MC, Hoes AW, Kors JA, et al. QTc dispersion predicts cardiac mortality in the elderly: the Rotterdam study. *Circulation.* 1998;97:467–472.
5. Zareba W, Moss AJ, Le Cessie S. Dispersion of ventricular repolarization and arrhythmic cardiac death in coronary artery disease. *Am J Cardiol.* 1994;74:550–553.
6. Glancy JM, Garratt CJ, Woods KL, et al. QT dispersion and mortality after myocardial infarction. *Lancet.* 1995;345:945–948.
7. Zaidi M, Roberts A, Fesler R, et al. Dispersion of ventricular repolarisation: a marker of ventricular arrhythmias in patients with previous myocardial infarction. *Heart.* 1997;78:371–375.
8. Dabrowski A, Kramarz E, Piotrowicz R. Dispersion of QT interval following ventricular premature beats and mortality after myocardial infarction. *Cardiology.* 1999;91:75–80.
9. Okin PM, Devereux RB, Howard BV, et al. Assessment of QT interval and QT dispersion for prediction of all-cause and cardiovascular mortality in American Indians. The strong heart study. *Circulation.* 2000;101:61–66.
10. Murakawa Y, Inoue H, Nozaki A, et al. Role of sympathovagal interaction in diurnal variation of QT interval. *Am J Cardiol.* 1992;39:339–343.
11. Ong JJC, Sarma JSM, Venkataraman K, et al. Circadian rhythmicity of heart rate and QTc interval in diabetic autonomic neuropathy: implications for the mechanism of sudden death. *Am Heart J.* 1993;125:744–752.
12. Molnar J, Zhang F, Weiss J, et al. Diurnal pattern of QTc interval: how long is prolonged? Possible relation to circadian triggers of cardiovascular events. *J Am Coll Cardiol.* 1996;27:76–83.
13. Ishida S, Nakagawa M, Fujino T, et al. Circadian variation of QT interval dispersion: correlation with heart rate variability. *J Electrocardiol.* 1997;30:205–210.
14. Nakagawa M, Takahashi N, Iwao T, et al. Evaluation of autonomic influences on QT dispersion using the head-up tilt test in healthy subjects. *PACE.* 1999;22:1158–1163.
15. Wagner KJ, Möllenberg O, Rentrop M, et al. Guide to anaesthetic selection for electroconvulsive therapy. *CNS Drugs.* 2005;19: 745–758.
16. McLaughlin NB, Campbell RWF, Murray A. Accuracy of four autonomic QT measurement techniques in cardiac patients and healthy subjects. *Heart.* 1996;76:422–426.
17. Xue Q, Reddy S. Algorithms for computerized QT analysis. *J Electrocardiol.* 1998:30(suppl):181–186.
18. Fisch C. Electrocardiography. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine.* 5th ed. Philadelphia: W.B. Saunders Company; 1997:114.
19. Rowlands DJ. Practical aids in ECG interpretation. In: Rowlands DJ, ed. *Clinical Electrocardiography.* Philadelphia: Lippincott Company; 1991:263–264.
20. Browne KF, Zipes DP, Heger SS, et al. Influence of the autonomic nervous system on the Q-T interval in man. *Am J Cardiol.* 1982;50:1099–1103.
21. Vieweg WVR, Wood MA. Tricyclic antidepressants, QT interval prolongation, and torsade de pointes. *Psychosomatics.* 2004;45:371–377.
22. Reilly JG, Ayis SA, Ferrier IN, et al. QTc-interval abnormalities and psychotropic drug therapy in psychiatric patients. *Lancet.* 2000;355:1048–1052.
23. Straus SMJM, Bleumink GS, Dieleman JP, et al. Antipsychotics and the risk of sudden cardiac death. *Arch Intern Med.* 2004;164: 1293–1297.
24. Rasmussen KG, Hooten WM, Dodd ML, et al. QTc dispersion on the baseline ECG predicts arrhythmias during electroconvulsive therapy. *Acta Cardiol.* 2007;62:345–347.
25. Saito S. Anesthesia management for electroconvulsive therapy: hemodynamic and respiratory management. *J Anesth.* 2005;19:142–149.
26. Kleinsasser A, Kuenszberg E, Loeckinger A, et al. Sevoflurane, but not propofol, significantly prolongs the Q-T interval. *Anesth Analg.* 2000;90:25–27.
27. Rasmussen KG, Laurila DR, Brady BM, et al. Anesthesia outcomes in a randomized double-blind trial of sevoflurane and thiopental for induction of general anesthesia in electroconvulsive therapy. *J ECT.* 2007;23:236–238.
28. Ramussen KG, Spackman TN, Hooten WM. The clinical utility of inhalation anesthesia with sevoflurane in electroconvulsive therapy. *J ECT.* 2005;21:239–242.
29. Hodgson RE, Dawson P, Hold AR, et al. Anaesthesia for