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# *Changes in the Skin Temperature of the Trunk and Their Relationship to Sympathetic Blockade during Spinal Anesthesia*

*Dermot P. Chamberlain, M.B., B.Chir., M.R.C.P.(UK), F.R.C.P.(C),**
*Beatrice D. L. Chamberlain, M.B., B.S., D.M.R.D.(Eng)†*

**Alterations in the skin temperature of the trunk during spinal anesthesia, using either tetracaine, 6 mg, or lidocaine, 50 mg, were monitored at intervals by means of a General Electric Spectrotherm 2000® Thermographic Imager and recorded on Polaroid® 107C photographic film. The upper level of sensory blockade was determined with each thermograph by recording the most cephalad dermatome at which analgesia to pinprick occurred. The upper limit of diminished sympathetic activity was assumed to be the most cephalad dermatome at which skin temperature elevation occurred. In all cases, the uppermost level of temperature elevation was cephalad to the upper limit of sensory blockade. Assuming that temperature elevation reflects diminished sympathetic activity, the mean sympathetic–sensory differential for lidocaine, 50 mg, was 6.00 (±SE 0.70) segments, and for tetracaine, 6 mg, was 6.70 (±SE 0.50) segments. Arrival of the temperature elevation "front" at the fourth thoracic dermatome and above was associated with decreases in mean arterial pressure. (Key words: Anesthetic techniques: spinal; sympathetic block. Measurement techniques: infrared thermography.**

SEVERAL STUDIES HAVE attempted to demonstrate sympathetic activity by measurement of skin temperature and capillary blood flow. Skin temperature has been measured with thermocouples,‡ electronic thermistor-thermometers,[1] electronic integrators,[2] liquid crystal thermography,[3] and infrared thermography.[4,‡] Blood flow has been assessed using washout techniques[5] and laser Doppler flowmetry.[1,6] Of these, infrared thermography has the advantages of being noninvasive, remote from the patient, and capable of producing multiple recordings at short time intervals. However, not all thermographic units have good resolution, and interpretation of thermographs where small variations in temperature occur is often subjective.

Greene[7] estimated the sympathetic sensory differential in spinal anesthesia using tetracaine by comparing the level of loss of sensation to pinprick with that of loss of temperature discrimination, on the basis that temperature sensation is carried by fibers of comparable size to the sympathetic fibers. The sympathetic–sensory differential assessed by this method was approximately two segments.

The Spectrotherm® Thermographic Imager (General Electric Corp.) has been used to monitor temperature changes noninvasively.[8] This type of imager can be used remote from the patient and can be adjusted to measure temperatures from 18–40° C in scan ranges from a 2° C scan up to a 16° C scan with a resolution of better than 0.1° C. Furthermore, the temperature pattern occurring along the reference line is plotted automatically by the imager, eliminating the subjective element in the interpretation of thermographs. We used this instrument to monitor the course of skin temperature change during spinal anesthesia.

## Methods

Twenty patients undergoing minor urologic procedures, who consented to spinal anesthesia and gave informed consent, were included in this study. Patients with documented neurologic disease were not studied. No premedication was given. Height, weight, and sex were noted.

The drugs lidocaine, 50 mg in 1 ml of 7.5% dextrose, and tetracaine, 6 mg in 1.2 ml of 5% dextrose, were randomly allocated such that ten patients received lidocaine and ten received tetracaine. The anesthetist assigned to the case performed the block, made all measurements of pulse and blood pressure, and determined the level of sensory analgesia to pinprick. The identity of the drug was not revealed until all observations had been completed.

The patient's trunk was left uncovered for at least 20 min prior to administration of the block. Oral temperatures were taken before the anesthetic was administered and at 10-min intervals thereafter for 30 min. Room temperature was constant at 22.5° C throughout the study.

An 18-gauge iv cannula was inserted into the left arm, and an intravenous infusion of 5% dextrose in lactated Ringer's solution was administered at a rate of 150 ml/h. Oxygen was given at a rate of 3 l per min *via* nasal cannula and respiration was monitored with a precordial stethoscope. The ECG was monitored using precordial leads, and the blood pressure was measured *via* a cuff on the arm using Korotkoff sounds.

The patient was placed supine with the right arm ab-

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* Assistant Professor of Anesthesiology.

† Volunteer Assistant Professor of Anesthesiology.

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Address reprint requests to Dermot Chamberlain, M.B.

‡ Gordh T: Analysis of the sensation of warmth in the lower extremities as the primary effect in spinal anesthesia. Regional Anesthesia 2:5–7, 1977.

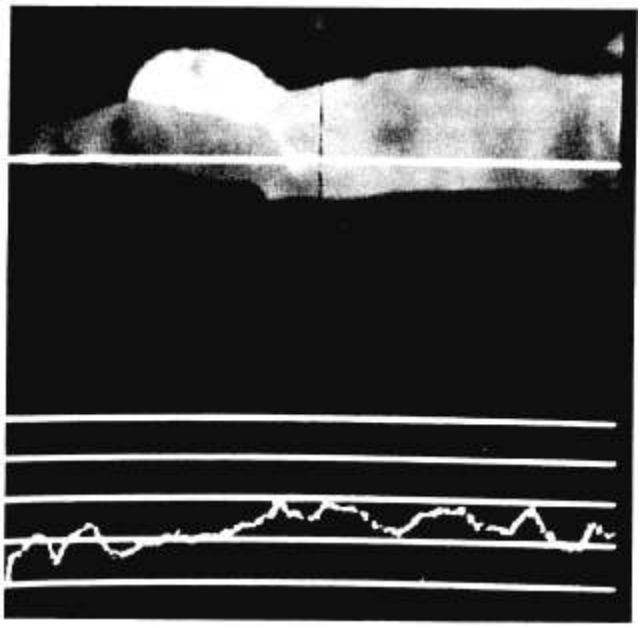

FIG. 1. Thermograph illustrating temperature pattern along midaxillary line (white reference line superimposed on patient). Lowermost calibration line is 32° C, and each of the four horizontal lines above this represents 1° C increments.

ducted to reveal the axilla and inner aspect of the upper arm. A control thermograph was taken of the right side of the trunk and upper arm with the temperature reference line set along the midaxillary line so that a graph of skin temperature from the dermatomes T-2 to L-1 was displayed. Spinal anesthesia was then performed with the patient in the left lateral position using a 22-gauge spinal needle inserted in the first interspace below the line connecting the iliac crests. A stop clock was started on completion of the injection, and the patient was repositioned in the "control" position.

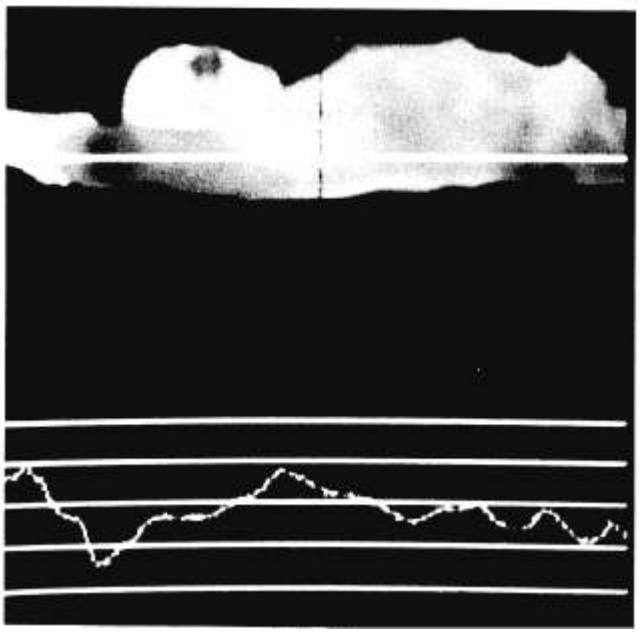

FIG. 2. Thermograph of same patient as in figure 1 28 min after subarachnoid administration of tetracaine, 6 mg, showing elevation of temperature of the trunk up to the axilla.

Mechanical stimulation of the skin along the right midaxillary line was avoided to reduce the possibility of local vasodilatory effects. Sensory level estimation was performed between the midline and the right anterior axillary line.

Hypotension (fall in blood pressure greater than 20%) was treated by administering iv fluids in 250 ml boluses or increments of ephedrine, 5 mg iv. Bradycardia (heart rate less than 50 beats/min) was treated with atropine, 0.2 mg iv, as indicated.

Thermographs were recorded on Polaroid® 107C film at a minimum of 5-min intervals except where surgical maneuvers rendered this impossible. The upper level of sensory blockade was determined each time a thermograph was taken and was recorded as the most cephalad dermatome at which analgesia to pinprick occurred. Recordings were continued for 30 min after the subarachnoid injection.

Changes in temperature along the reference line with respect to the control thermograph were noted according to the dermatome level. The oral temperatures were plotted for each case so that any decrease from the control level could be determined and added to the measured skin temperature value at any given time, thus compensating for a cooling effect. In this way, temperature changes occurring from T-1 to L-2 for 30 min following subarachnoid injection, together with the corresponding levels of sensory analgesia, could be obtained.

Differences between the tetracaine and lidocaine groups, *i.e.*, sensory level, time to attain maximum level of temperature change, sympathetic–sensory differential, and time of onset of maximum sensory blockade, were tested for significance by means of the Mann-Whitney U test.

Approval for this study was obtained from the Committee on Human Research of the University of Cincinnati.

## Results

Figures 1 and 2 are illustrative of the material from which our data and interpretations are drawn. The supine patient can be seen, the tone of the thermograph varying from black (cold) to white (hot). The white horizontal line superimposed on the patient's thermograph is a temperature reference line positioned at the midaxillary level by the investigator. The set of five horizontal white lines in the lower half of the illustrations are the temperature

TABLE 1. Mean Maximum Temperature Elevation, both Observed and Corrected for Cooling, for Lidocaine and Tetracaine Groups

| Dermatome | Mean Maximum Temperature Elevation (° C) | | | |
|---|---|---|---|---|
| | Lidocaine (±SE) | | Tetracaine (±SE) | |
| | Observed | Corrected for Cooling | Observed | Corrected for Cooling |
| T-1 | 0.93 ± 0.20 | 1.63 ± 0.30 | 1.28 ± 0.14 | 1.73 ± 0.21 |
| T-2 | 0.69 ± 0.14 | 1.24 ± 0.21 | 0.84 ± 0.11 | 1.47 ± 0.16 |
| T-3 | 0.60 ± 0.10 | 1.12 ± 0.19 | 0.90 ± 0.22 | 1.62 ± 0.21 |
| T-4 | 0.62 ± 0.15 | 1.12 ± 0.20 | 0.74 ± 0.20 | 1.17 ± 0.21 |
| T-5 | 0.49 ± 0.12 | 0.84 ± 0.18 | 0.68 ± 0.19 | 0.90 ± 0.26 |
| T-6 | 0.23 ± 0.08 | 0.82 ± 0.21 | 0.48 ± 0.15 | 0.88 ± 0.24 |
| T-7 | 0.28 ± 0.07 | 0.85 ± 0.18 | 0.38 ± 0.13 | 0.96 ± 0.25 |
| T-8 | 0.17 ± 0.06 | 0.76 ± 0.16 | 0.42 ± 0.14 | 0.86 ± 0.26 |
| T-9 | 0.19 ± 0.06 | 0.77 ± 0.16 | 0.47 ± 0.16 | 0.89 ± 0.28 |
| T-10 | 0.23 ± 0.10 | 0.79 ± 0.16 | 0.40 ± 0.14 | 0.95 ± 0.28 |
| T-11 | 0.29 ± 0.09 | 0.74 ± 0.16 | 0.46 ± 0.15 | 0.81 ± 0.30 |
| T-12 | 0.32 ± 0.11 | 0.82 ± 0.19 | 0.54 ± 0.17 | 0.99 ± 0.32 |
| L-1 | 0.43 ± 0.13 | 0.82 ± 0.27 | 0.54 ± 0.15 | 0.89 ± 0.34 |
| L-2 | 0.67 ± 0.22 | 0.90 ± 0.35 | 0.60 ± 0.18 | 1.50 ± 0.10 |

Mean cooling (oral temperature) for all cases: 0.75° C ± SE 0.11° C.

TABLE 2. Time from Completion of Subarachnoid Injection for the Temperature Elevation Wavefront to Reach T-1 or Uppermost Level, with the Corresponding Sensory Blockade Level Achieved

| | Lidocaine | | | | Tetracaine | | | |
|---|---|---|---|---|---|---|---|---|
| Case | Time (min) | Temperature Level | Sensory Level* | | Case | Time (min) | Temperature Level | Sensory Level* |
| 1 | 17.5 | T-1 | T-7 | (6) | 1 | 18 | T-1 | T-7 (7) |
| 2 | 7.5 | T-1 | T-11 | (5) | 2 | 14 | T-1 | T-6 (6) |
| 3 | 25 | T-1 | T-9 | (9) | 3 | 25 | T-1 | T-6 (6) |
| 4 | 27 | T-2 | T-10 | (9) | 4 | 30 | T-1 | T-10 (10) |
| 5 | 18 | T-1 | T-6 | (4) | 5 | 13.5 | T-1 | T-8 (8) |
| 6 | 27 | T-1 | T-7 | (7) | 6 | 10 | T-1 | T-7 (4) |
| 7 | 6 | T-1 | T-5 | (3) | 7 | 15 | T-1 | T-10 (10) |
| 8 | 30 | T-1 | T-8 | (8) | 8 | 18 | T-1 | T-9 (9) |
| 9 | 25 | T-5 | T-6 | (6) | 9 | 5.5 | T-1 | T-8 (8) |
| 10 | 30 | T-1 | T-6 | (6) | 10 | 30 | T-1 | T-6 (6) |

End-point: Time at which temperature elevation "front" reaches most cephalad dermatome up to T-1.

* Numbers in parentheses indicate sensory level at 30 min from completion of injection.

calibration lines. In these examples the lowest line is set at 32° C and the scan range is set at 4° C. The vertical distance between each horizontal line therefore represents 1° C. Both thermographs are of the same patient, the second (fig. 2) having been taken 28 min following subarachnoid injection of tetracaine, 6 mg. Comparing the temperature plots that relate to the skin temperature along the midaxillary reference line, the elevated trunk temperature is clearly seen. In the axillary region the skin temperature has risen more than 1° C. It should be noted that the elbow in both thermographs remains "cold," indicating that although sympathetic block has caused an increase in the skin temperature of the trunk, the intensity of block is not sufficient to cause warming of the arm. During the 30-min study period this patient's oral temperature fell 1° C.

The temperature changes at the dermatome levels T-1 to L-2 were tabulated and, after correction for "core" cooling as described in "Methods," the most cephalad dermatome at which temperature elevation occurred was recorded as the upper extent of temperature change. The mean maximum observed and corrected temperature elevations encountered at each dermatome are tabulated in table 1. As can be seen, even without allowing for "core" cooling, temperature rises were observed. The smallest rises were observed in the abdominal region. This phenomenon may be due to the possibility that sympathetic block may produce greater net increases in flow in the higher dermatomes.

The times at which the temperature elevation "front" reached the most cephalad dermatome up to T-1 are shown in table 2. All patients receiving tetracaine eventually showed temperature elevation up to T-1, and eight patients in the lidocaine group showed a rise to this level. Table 3 shows the time intervals after block at which these maximal levels were attained.

The patients' blood pressures were recorded every 5 min, and the relationship between percentage change in mean arterial blood pressure relative to the control value and the upper limit of temperature rise over the thoracic dermatomes were studied. Arrival of the temperature elevation front at the fourth thoracic dermatome for both the lidocaine and tetracaine groups was consistently associated with a drop in mean arterial pressure. Four patients in the lidocaine group and three in the tetracaine group required treatment for hypotension, and one of these in the lidocaine group also had bradycardia that was treated with atropine. In these patients such therapy was administered after temperature elevation had reached T-1 and hence did not affect the results.

Figure 3 shows the mean sensory levels and corre-

TABLE 3. Percentage of Patients Showing Temperature Elevation at T-1 at Different Time Intervals Following Subarachnoid Injection

| Time after Subarachnoid Injection (min) | Percentage of Patients Showing Temperature Rise at T-1 Level | |
|---|---|---|
| | Lidocaine | Tetracaine |
| 0–5 | 0 | 0 |
| 6–10 | 20 | 20 |
| 11–15 | 20 | 40 |
| 16–20 | 40 | 60 |
| 21–25 | 80 | 90 |
| 26–30 | 80 | 100 |

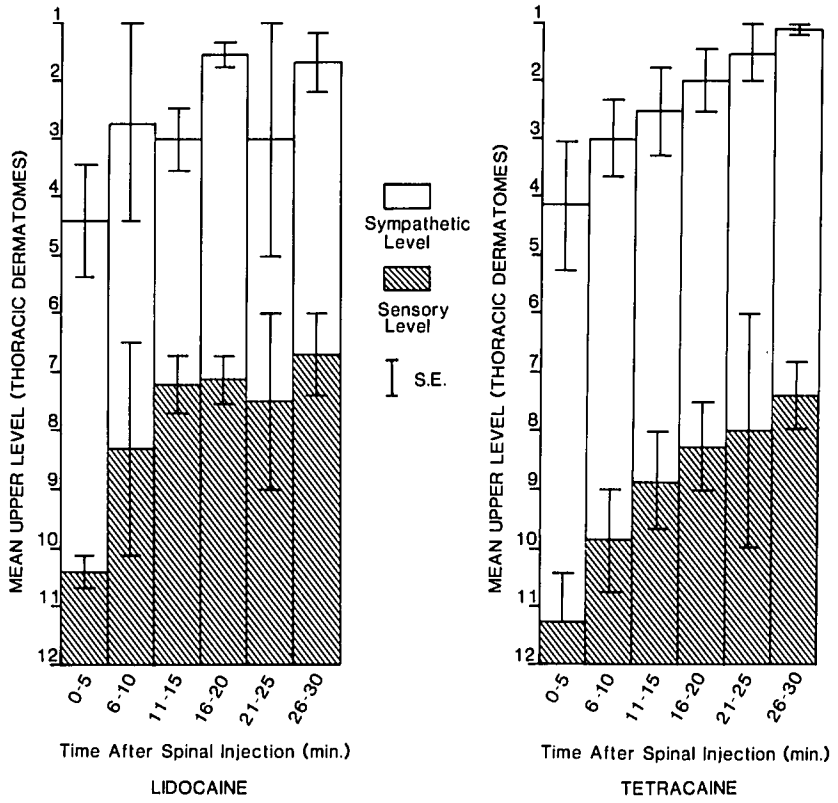


FIG. 3. Mean sensory levels and corresponding upper limits of temperature elevation expressed in dermatomes over 5-min intervals following the subarachnoid injections up to 30 min.

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Our results are summarized in table 4. The sympathetic–sensory differential is taken to be the segmental difference between the temperature elevation "front" and the upper level of sensory analgesia. No significant difference was found between the groups on applying the Mann-Whitney U test.

TABLE 4. Summary of Findings

| | Lidocaine (±SE) | Tetracaine (±SE) |
|---|---|---|
| Mean maximum sensory level* (dermatomes) | T-6.3 ± 0.63 | T-7.50 ± 0.56 |
| Mean time to attain maximum sensory level* (min) | 17.23 ± 3.20 | 15.45 ± 2.16 |
| Mean time to attain maximum level temperature rise* (min) | 19.70 ± 2.68 | 17.40 ± 2.37 |
| Mean sympathetic–sensory differential*† (dermatomes) | 6.00 ± 0.70 | 6.70 ± 0.50 |

* No significant difference between groups on applying Mann-Whitney U test.

† The assumption that temperature elevation reflects diminished sympathetic activity is made.

Room temperature remained constant at 22.5° C throughout the study period.

## Discussion

We have used a highly sensitive instrument to detect the dynamic pattern of skin temperature changes during spinal anesthesia. The study was designed to reduce to a minimum causes of increased skin temperature other than sympathetic nerve blockade. Arrival of the wavefront of skin temperature increase at the T-4 dermatome was consistently associated with a decrease in mean arterial pressure, supporting the hypothesis that the most cephalad extent of skin temperature elevation corresponds with the upper limit of diminished sympathetic activity, and is in agreement with the view that skin temperature increase is a useful indicator of sympathetic blockade. We can make no assumption regarding the intensity of sympathetic blockade, and in view of the fact that sympathetic fibers leaving the spinal cord travel up or down three or more segments before entering the sympathetic ganglia, it is unlikely that the level of the temperature elevation wavefront reflects total sympathetic blockade. Further support of the hypothesis of the close relationship between skin temperature elevation and sympathetic blockade is lent by the fact that temperature elevation always preceded the upper limit of sensory blockade and had a similar pattern of onset.

Recent work using thermography by Bengtsson[4] has thrown doubt on the validity of relating skin temperature elevation to sympathetic blockade. The AGA Thermovision® used can detect temperature differences of 0.1° C, but the display monitor can only depict temperature differences in a five-step gray scale for a given scan range. According to Normell,[9] the AGA Thermovision® must detect temperature differences equal or greater than 1° C before the result can be regarded as significant. In Bengtsson's series the patients received premedication, their central temperatures were not monitored, and there is no indication as to whether intraoperative drugs were administered. Thus, it is not possible to determine whether skin temperature changes due to sympathetic blockade were masked by pharmacologic agents or other factors. The patients in our study received no premedication or drugs known to affect temperature, such as narcotics and phenothiazines.

Studies by Bengtsson *et al.*[1,4] of skin blood flow using a laser Doppler flowmeter during spinal anesthesia suggest decreased rather than increased cutaneous flow. However, the laser Doppler used measured flow at a depth of 0.5 to 1 mm, which may not be a representative depth, and the instrument itself measures flow by measurement of the velocity of red blood cells flowing through a capillary. However, the velocity of red blood cell movement is only directly proportional to flow if the mean cross-sectional area of the capillaries remains constant.[6] Because sympathetic blockade causes vasodilatation through removal of the vasoconstrictor tone, the laser Dopple flowmeter is unlikely to be an accurate indicator of sympathetic activity unless simultaneous measurements of the diameter of the vessel are performed. Indeed, in some cases, increased flow was associated with a drop in temperature at the same dermatome level as measured with a thermistor temperature probe.

Our results have shown, as did Greene's[7,10] that a segmental sympathetic–sensory differential during spinal anesthesia and tetracaine exists and that the level of sympathetic blockade lies cephalad to that of sensory blockade. Using loss of temperature discrimination as the indicator of sympathetic blockade, Greene found that the mean sympathetic–sensory differential for tetracaine was two segments. Greene has stated that the cardiovascular changes (hypotension and bradycardia) observed during spinal anesthesia in the normovolemic, supine subject with sensory block level at or below T-6 could not be explained in terms of blockade of the cardiac accelerator fibers. He has postulated[10] that these effects are due to activation of the intracardiac stretch receptors, located primarily in the right atrium, by the decreased venous return caused by sympathetic blockade. Our study, however, shows that diminished sympathetic activity at and above the T-4 level occurs even with sensory blockade below T-6. We found the mean sympathetic–sensory differential to be greater than six segments for tetracaine, and we therefore conclude that blockade of the cardiac accelerator fibers may be a major contributing factor in the production of hypotension and bradycardia, even when the sensory blockade level is at or below the T-6 level.

We cannot state that our results are at variance with Greene's as temperature elevation indicates diminished sympathetic activity, and we cannot determine at what level total blockade occurs. We have observed temperature elevation at the T-1 level in patients where the forearm and hand are cold. We therefore conclude that sympathetic blockade up to the T-1 level is only partial. It is possible that Greene's method detects the level of total sympathetic blockade and that our method demonstrates the upper limit of partial blockade, and the four-segment difference between our results and Greene's could be explained in anatomic terms, for it is well recognized that overlap of three or more segments occurs in sympathetic innervation.

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## References

1. Bengtsson M, Nilsson GE, Lofstrom JB: The effect of spinal analgesia on skin blood flow, evaluated by laser Doppler flowmetry. Acta Anaesthesiol Scand 27:206–210, 1983
2. Delius W, Hagbarth KE, Hongell A, Walling BG: Maneuvers affecting sympathetic outflow in human skin nerves. Acta Physiol Scand 84:177–186, 1972
3. Pochaczevsky R: The value of liquid crystal thermography in the diagnosis of spinal root compression syndromes. Orthop Clin North Am 14:271–288, 1983
4. Bengtsson M: Changes in skin blood flow and temperature during spinal analgesia evaluated by laser Doppler flowmetry and infrared thermography. Acta Anaesthesiol Scand 28:625–630, 1984
5. Skagen K, Haxholdt O, Henriksen O, Dyrberg V: Effect of spinal sympathetic blockade upon postural changes of blood flow in human peripheral tissues. Acta Physiol Scand 114:165–170, 1982
6. Holloway GA, Watkins DW: Laser Doppler measurement of cutaneous blood flow. J Invest Dermatol 69:306–309, 1977
7. Greene NM: The area of differential block during spinal anesthesia with hyperbaric tetracaine. ANESTHESIOLOGY 19:45–50, 1958
8. Abramson PR, Pearsall EH: Pectoral changes during the sexual response cycle: A thermographic analysis. Arch Sex Behav 12: 357–368, 1983
9. Normell LA: Recording of normal and impaired cutaneous thermoregulatory vasomotor responses by infrared thermography: A methodological study. Scand J Clin Lab Invest 138(Suppl 33):3–24, 1974
10. Greene NM: Preganglionic sympathetic blockade in man: A study of spinal anesthesia. Acta Anaesthesiol Scand 25:463–469, 1981