SWEATING AND ANESTHESIA
A CONSIDERATION OF CAUSES AND EFFECTS

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Sweating in patients undergoing surgical procedures is seen by most anesthetists from time to time. In this discussion an attempt will be made to correlate the literature and clinical observations and bring into focus some facts and theories which might be of interest to the practicing anesthetist. Before considering clinical applications, however, a brief summary of the anatomy and physiology of thermal control would be pertinent.

ANATOMY OF SWEAT GLAND INNERVATION

A “thermal center” is present in the hypothalamus and the preoptic area. There is also some evidence of cortical activity affecting certain types of sweating, but no true center has been demonstrated. “Local” centers are present in the thoracic and lumbar regions of the spinal cord, as may be suspected by the fact that sweating still occurs, though to a less extent, in the lower limbs even after transection of the spinal cord.1 The descending tracts in the spinal cord and efferent fibers to the sweat glands are anatomically part of the sympathetic outflow of the autonomic nervous system.2 From the hypothalamus a neuronal tract passes through the medulla and descends to the spinal cord terminating in the intermediolateral horn at various levels between the first thoracic to the second or third lumbar segments. From each segment the efferent fiber leaves the cord in the anterior nerve root. The fibers form part of the mixed spinal nerve and anterior primary ramus and then passing through the white ramus communicans join the corresponding paravertebral ganglion of the sympathetic chain.3 Most of these fibers do not relay in the ganglia of the sympathetic chain, or in any peripheral ganglia, but pass directly to the sweat gland receptor where, as with all preganglionic fibers, acetylcholine is the excitor substance released. There is some evidence, however, to suggest that certain of the fibers do relay in the sympathetic chain, and postganglionic fibers which then pass to the sweat glands liberate adrenaline as the excitor substance.4,5 This may account, in part at least, for the fact that sweating can occur following both sympathetic and parasympathetic activity as a result of adrenaline circulating in the blood stream and stimulating the sweat glands directly.

Afferent impulses for temperature sensation arising from thermal nerve endings pass in the peripheral sensory nerves which enter the spinal cord via the posterior root and cross at once to the opposite lateral spinothalamic tract (fig. 1). This tract passes up the spinal cord, the fibers conveying temperature impulses lying lateral and superficial in the medulla and passing more medially as they enter the pons. The fibers terminate in the thalamus. Further neurones carrying impulses destined for consciousness pass from here to the sensory cortex.

PHYSIOLOGY OF SWEATING

Thermal Sweating. The primary purpose of sweating is to lose heat by evaporation. Therefore, apart from the different causes of sweating due to autonomic activity to which reference will be made later, sweating occurs as a response to a rise in temperature. Sweating in response to external heat occurs reflexly before a rise in temperature in the body as a whole can be detected, thus sweating acts as a preventive against heat accumulation. It is, in fact, a more efficient measure against heat accumulation than as a means of reducing temperature once it has been raised.6 The stimulus to the hypothalamic center is probably twofold. Firstly, it is stimulated by nervous impulses from the nerves carrying temperature impulses; secondly, it may be directly affected by the temperature of the
blood passing over it. Thermal sweating tends to be generalized all over the body. Indeed, if one area of the body is heated the whole body sweats, although sweating will be more profuse in the heated area.

When the surrounding temperature approaches or exceeds the skin temperature, the loss of heat by radiation or convection is reduced. In fact, the body may actually gain heat as witness the act of taking a hot bath where the area available for sweating is usually confined to the head and neck. Temperature regulation by sweating has priority over water and electrolyte balance.\(^a\) Sweating will continue even though it causes severe dehydration and salt loss. Only when the circulation fails will sweating cease.

Sweating is not seen in newborn infants, but it occurs within the first few days of life. Until puberty children sweat more readily than adults and as a generalization males sweat more than females.

**Autonomic Sweating.** Under this heading may be classified sweating which occurs from stimuli other than thermal. The autonomic activity may be either sympathetic through the
TABLE 1

<table>
<thead>
<tr>
<th>Thermal</th>
<th>Autonomic</th>
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<tr>
<td>Skin</td>
<td>Hot</td>
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<tr>
<td></td>
<td>Generalized</td>
</tr>
<tr>
<td>Onset</td>
<td>Delayed</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Raised, (e.g., after exercise)</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>Raised, (e.g., fever)</td>
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<tr>
<td>Body Temperature</td>
<td>May be raised (if heat stimulus is prolonged)</td>
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mediation of adrenaline and noradrenaline, or parasympathetic through the mediation of acetylcholine.8

Although the innervation of the sweat glands is primarily cholinergic, there is no doubt that adrenaline and noradrenaline have a role in the production of sweating.5,9 If injected intradermally both adrenaline and noradrenaline will cause local sweating by direct action on the sweat glands, and apart from this, as mentioned previously, there is some physiological evidence that the innervation of some of the sweat glands is adrenergic.10 The two drugs are about equal in action, and sweating so produced is not inhibited by atropine but is inhibited by adrenergic blocking agents, e.g., dibenamine. It may be that in certain conditions such as exercise, where the sympathetic system has priority, circulating adrenaline and noradrenaline exert a direct effect on the sweat glands and may add to the effect produced by the cholinergic nerves.

Whether a particular individual will react with a sympathetic or parasympathetic response cannot be predetermined with any accuracy. The overtalkative patient about to undergo some surgical procedure may try to conceal his nervousness by his verbosity. He is likely, however, to show profuse sweating in the palms of the hands and the axillae. His pulse rate and blood pressure may also be raised. The patient is showing a typical sympathetic response. Conversely an individual who witnesses a traffic accident, or even a cut finger, may experience a profound fall in blood pressure and pulse rate, break out in a generalized “cold sweat,” and may even lose consciousness. This is the parasympathetic response.

Such autonomic sweating can occur under general anesthesia and the response may be either sympathetic or parasympathetic. Further reference will be made to this later. The three types of sweating are summarized in table 1.

EARLY SIGNS AND COMPLICATIONS ASSOCIATED WITH SWEATING DURING SURGERY

Early signs associated with thermal sweating may be:

(a) Rise in temperature—occurs after sweating has been established.
(b) Rising pulse rate, in absence of any other obvious cause.
(c) Alteration in blood pressure. A fall may be seen due to vasodilation, particularly if a head-up position is adopted. Conversely in early stages of carbon dioxide retention a rising blood pressure may be associated with sweating.
(d) There may be an increase in respiratory rate. This is consequent to hyperpyrexia and is not related to surgical trauma since it does not cease even if the stimulus of trauma is removed. An excessive rise in temperature will cause a depression of the medullary centers and eventual inhibition of respiration.
SWEATING AND ANESTHESIA

If sweating is extensive and prolonged there is excessive fluid loss, both of water and electrolytes, and almost all the complications of sweating can be related to this fact. Excessive fluid loss leads to hemoconcentration with consequent increase in work for the heart. Cardiac output is reduced and if dehydration is severe and prolonged, cardiac failure will supervene. Conditions severe enough to give rise to cardiac failure are seldom seen clinically, but could occur in severe climatic conditions.

Factors Modifying a Sweat Response During Surgery

If a deep general anesthetic is administered, the heat control mechanisms are depressed and the subject tends to adopt the temperature of his surroundings. This is particularly true of children. However, most general anesthetics today are kept on a light plane and relaxation obtained when necessary by use of relaxant drugs. Under such conditions there is only moderate depression of the temperature regulating centers. Indeed, with adequate premedication, good ventilation and no muscular straining, heat production may approach basal levels.

Apprehensive patients may be observed to be sweating in the induction room. Apart from possibly implying inadequate preoperative sedation, this type of sweating is of little significance in itself. It is, however, worthwhile emphasizing that the anesthetist should ensure himself that the sweating is emotional and not, for example, the “cold sweat” of a shocked patient or an oversedated one.

Surgical stimulation in light planes of anesthesia may produce a sympathetic response with a rise in pulse rate and blood pressure with or without sweating. There can be severe sweating in the removal of a pheochromocytoma due to liberated adrenaline. Conversely certain types of surgical trauma, hemorrhage, or pain are more likely to produce a parasympathetic response. This is clinically recognizable as a “cold sweat” and is usually but not always associated with a bradycardia and reduced cardiac output consequent upon vagal activity. The conscious patient under regional anesthesia may feel nauseated and faint and the skin feel cold and clammy. In this circumstance vagal response is of more serious import than loss of fluid due to sweating. Certain types of surgical manipulation are more likely to produce this type of reaction, e.g., traction on the mesentery or esophagus, pressure on the roots of the lungs, stretching the uterine cervical canal. Some anesthetists have noted that by blocking the splanchnic nerves with local anesthetic some of these responses may be blocked.

Anesthetic Drugs. Ether, being a sympathomimetic drug, may initiate sweating in light anesthesia. In light levels of anesthesia cyclopropane stimulates the sympathetic system and there is an increased liberation of catecholamines. Yet sweating is rare with cyclopropane, and instead of the tachycardia that one might expect as a result of sympathetic activity, it is more usual to find a bradycardia. The explanation lies in the fact that cyclopropane stimulates both sympathetic and parasympathetic systems but the latter predominates. Halothane depresses sympathetic activity but has little effect on the parasympathetic. Thus the bradycardia seen with halothane is due to activity of an unopposed parasympathetic system, and sweating is seldom seen. Sweating, due to parasympathetic activity, is usually the result of reflex activity. In contrast, sweating seen as a result of the effects of a general anesthetic, is due to central stimulation. Intravenous anesthetics, such as thiopental, will not produce sweating unless given rapidly or in high doses when the myocardium is depressed and a state of shock is produced.

Carbon dioxide retention will cause a rise in blood pressure, both via the chemoreceptors and by direct effect on the vasomotor center. There is also a direct action on the peripheral arterioles producing vasodilatation. The skin temperature rises and sweating may occur. Such a situation can arise insidiously in mildly underventilated patients.

Oxygen Lack. Hypoxia causes cyanosis, an initial rise in blood pressure and sweating. If continued the circulation fails and the skin becomes cold, pale and clammy. In this instance there is first a sympathetic response and then a parasympathetic. Sweating occurring when nitrous and oxygen are the sole anesthetic agents may be due to a combination of mild hypoxia and sympathetic stimulation.
Hypoglycemia. Sweating may also be seen in patients who are hypoglycemic. Not only may this occur in diabetic patients whose blood sugar control has become deranged, but may occasionally be seen in normal patients when due to surgery they may have missed one or more meals. There may be an associated tachycardia but the blood pressure is usually normal or slightly raised.

Temperature, humidity and ventilation will affect the formation and evaporation of sweat. In an operating room with air conditioning the usual limits of temperature are 68-72 F. and humidity 52-60 per cent. Under such conditions sweating from these factors should be minimal.

Drapes and rubber sheeting, although contributing to the sterility of the operating field, may seriously impede the evaporation of sweat, and the patient's temperature may rise. Nowhere is this more true than when dealing with children.

Paradoxically, a patient's temperature may rise to dangerous levels, and yet no sweating occurs. Such a situation can arise in patients who are either unduly sensitive to a usual clinical dose, or else have been given an excessive amount of atropine or scopolamine. Reduced doses should be given to patients with hyperpyrexia, and this is particularly true in children who exhibit a high fever much more readily than an adult. Rarely one may encounter a patient with congenital absence of sweat glands and the temperature in these persons can rise to dangerous levels after atropine and scopolamine.

Conclusions

If an anesthetist observes a patient sweating during anesthesia or operation, he should consider the following:

(1) Prevention being better than cure, it is well to have a saline or dextrose and water transfusion ready unless this is contraindicated because of disease or surgical techniques. Temperature recordings are useful in children and during long operations.

(2) If sweating is first observed in the induction room, the anesthetist should assure himself that this is only emotional.

(3) If sweating is of the parasympathetic variety he should check the pulse rate and blood pressure and inform the surgeon. A falling blood pressure associated with sweating is of more serious import than a rise in pressure accompanied by sweating. The cause may be obvious, but if not, cessation of surgery for a few minutes, may improve the condition. Vasoressors may be required for profound fall in blood pressure, or blood transfusion for hemorrhage. Raising the legs will aid venous return and improve cardiac output.

(4) Excessive drapes and rubber sheeting should be avoided, or ventilation under the drapes should be provided. This is a common cause of thermal sweating.

(5) If surgical stimulation is considered the cause of sweating, increasing the analgesia may block the response. This may be attained by deepening the inhalation anesthetic, the addition of anesthetic drugs intravenously or by the use of local or topical anesthetics—direct nerve blocks or instillation of local anesthetic into the peritoneal cavity. Up to 50 ml. of 1 per cent lidocaine may be used in adults with safety for this latter procedure.

(6) Care should be taken to ensure adequate pulmonary ventilation. Use of the closed circuit combined with adequate ventilation will effectively prevent carbon dioxide retention. If an open circuit is used, the gas flow must at least equal the patient's minute volume and the tidal exchange must be adequate. The use of nonreturn valves will lessen the chances of carbon dioxide retention.

(7) Hypoglycemia must be considered. If this is the cause, intravenous dextrose will rapidly relieve the condition.

(8) Patients should not exhibit sweating while undergoing surgery, and if it occurs, the anesthetist should make every attempt to find and eradicate the cause.

REFERENCES
PHENAZOCINE The effectiveness of a new potent synthetic analgesic narcotic of the benzomorphan series has been studied in a series of 778 surgical patients. The drug was given for purposes of preanesthetic medication, as an anesthetic adjuvant, for relief of postanesthetic restlessness and pain, and for relief of postoperative pain on the ward. The drug was found to be a potent narcotic with a rapid onset of action with minimal side effects. The duration of action was approximately equal to that of morphine. The relative potency to morphine is considered to be 1 mg. of phеназocine to 4 or 5 mg. of morphine. There appears to be a synergistic effect between phеназocine and other depressants. The undesirable side effects of phеназocine were those of all other narcotics. However, the incidence of side actions appeared smaller than with morphine, meperidine and allied drugs. Evidence of circula-

tory and respiratory depression was observed, but the incidence of such reactions was small. (Precosnik, S. J., and Eckenhoff, J. E.: Use of Phenazocine (Prainodol) in Surgical Patients, Surg. Gyneec. & Obstet. 110: 669 (June) 1960.)

PAIN CONTROL Ninety-six postoperative patients were given doses of 5, 10, and 20 mg. of pimino-nide · (Alvodine) or 10 mg. of morphine sulfate for relief of pain. The results were evaluated to determine the relative potency of pimino-nide in relation to morphine. A dose of 5 mg. of pimino-nide was less effective and 10 mg. of pimino-nide was more effective than a 10-mg. dose of morphine sulfate. Two episodes of vomiting occurred in the 19 patients given 20 mg. of pimino-nide. (De-Kornfield, T. J., and Lasagna, L.: Analgesic Potency of Pimino-nide (Alcodine), J. Chronic Dis., 12: 252 (Aug.) 1960.)