Autopsy was performed 10 hours after death. The 2,260 g. liver, of hobnail appearance, showed rare regenerative lobules of 1–4 mm. diameter, between large amounts of scar tissue; multiple microscopic slides confirmed the diagnosis of advanced generalized Laennec's cirrhosis, with a profusion of connective tissue, rich in biliary ducts, many of them dilated and containing a deeply stained inspissated bile. The parenchymal lobules, rare and of irregular size, contained normal cells, except for a few deeply bile stained or edematous areas; no cell showed necrosis or fatty metamorphosis. The kidneys were normal. The esophageal walls disclosed extensive ulcerations where the mucosa had been replaced by a thick necrotic membrane infiltrated with neutrophiles. The veins were very dilated. The gastrointestinal tract was filled with about two liters of bright red blood; the gastric mucosa showed innumerable pinpoint ulcerations. The epidural mass resected at operation was malignant and probably metastatic, but the primary lesion could not be found.

It is impossible to elicit the precipitating factor which led to the fatal hepatic failure. The time of onset of symptoms, however, points toward anesthesia rather than blood, antibiotics or another drug. Halothane, and its hypotensive effect on a probably hypovolemic patient is a prime suspect. The absence of zonal necrosis and fatty changes is worth emphasizing. In the 16 fatal cases reviewed by Blackburn (ANESTHESIOLOGY **25**: 270, 1964) and two others reported by Burns (Brit. Med. J. **2**: 483, 1957) and Chamberlain (Brit. Med. J. **1**: 1524, 1963), severe necrosis, usually with fatty changes were repeatedly present. Did the rapid evolution bring biochemical failure before the appearance of any morphological lesions? Or was the precipitating stimulus too small to cause pathological changes, while sufficient to produce functional failure in a diseased liver? This must remain speculative, but it suggests that severe biochemical damage may occur without histological changes and that liver biopsy may be misleading. Indeed, careful review of the chloroform literature discloses numerous fatal cases with minimal microscopic lesions, in spite of the insistence of many textbooks to describe a "classical central necrosis."

It is also possible that regenerative liver cells, although functionally sensitive, offer a morphological resistance to halothane. This abnormal response has been described with chloroform (McNider, J. Pharm. Exp. Ther. **59**: 393, 1937).

Postoperative (Pressure) Alopecia

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Abel and Lewis[1] reported 8 cases of a new type of alopecia, occurring only in women who had undergone prolonged gynecological operations. The alopecia always appeared over the vertex or the upper occiput. In 5 of the 8 patients there was pain, swelling, or serous discharge from the affected scalp within 72 hours after the operation. Hair loss began is this affected area in 3 to 28 days. It developed into a patch of alopecia, usually total, 2 to 8 cm. in diameter, resembling alopecia areata. By the time the hair loss occurred, the scalp appeared normal. Complete regrowth of hair occurred in all cases, usually starting within 90 days. Histopathological studies of the scalp in 6 cases showed the cardinal feature to be an obliterative vasculitis involving the vessels of the deep cutis and the fat. Two cases showed moderate panniculitis and two cases showed a mild perivascular lymphocytic infiltrate. Thomson and Estrellado[2] recently reported a series of three cases

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TABLE 1. Summary of Eight Cases of Postoperative (Pressure) Alopecia

Case	Description of Patient	Operation and Duration	Postoperative Scalp Symptoms	Number of Days Postoperatively Hair Loss First Noted	Position of Alopecia	Area of Alopecia (cm.)	Regrowth
1	34 year old Negro man.	7 hour gastrectomy.	None	10	Upper central vertex.	3 × 3	Complete in 2 months.
2	39 year old white woman.	4¾ hour hysterectomy.	Severe pain and tenderness over upper occiput for 3 weeks. Serum oozed from this area for several days.	21	Upper central occiput (Same area that was tender).	4 × 2	Complete in 3 months.
3	8 year old white male mongolian idiot.	5½ hour ureteroneocystostomy and ureteroplasty.	None.	15	Upper occiput.	5 × 2	Complete in 7 weeks.
4	40 year old white woman.	4¾ hour hysterectomy.	None.	23	Right upper occiput.	2.5 × 1.5	Complete in 3 months.
5	40 year old white woman.	4 hour hysterectomy.	Pain and tenderness over lower central vertex 24 hours postoperatively. Yellow crusting and swelling noted 5 days later.	12	Lower central vertex (The same area that was symptomatic).	1 × 3	Complete in 11 weeks.
6	35 year old white woman who was in deep coma for 48 hours after a suicide attempt.	—	None	26	Central vertex.	5.5 × 3	Early regrowth present at 45 days.
7	7 year old white man.	5½ hour right frontal craniotomy.	None	12	Left occiput.	4 × 4	Complete at 12 weeks.
8	36 year old white man.	7 hour total colectomy.	Crusting over lower vertex. When crusts were removed 14 days postoperatively, hair came out.	14	Lower central vertex.	8 × 4	Early regrowth at 6 weeks.

of what is apparently postoperative (pressure) alopecia occurring after open heart surgery.

Animal experiments, using steady prolonged pressure applied to the skulls of anesthetized cats, reproduced this condition. The pressure was of the same magnitude as that which the recumbent human head generates on its scalp because of its own weight.[1] Three out of four cats developed a patch of alopecia in the pressure area. Histopathological sections showed severe vascular damage.

Observations on New Cases

Since publication of the article on postoperative (pressure) alopecia,[1] 8 additional cases (table 1) have been observed which shed further light on this intriguing and preventable type of baldness.

Comment

These cases show that the condition may occur at any age, in either sex. The instance that occurred following coma indicates that while anesthesia is the most common preceding factor, it is not the only factor. The prime factor in the etiology of postoperative (pressure) alopecia appears to be a prolonged period of profound unconsciousness. Even though the patient's head is well cushioned (2 inches foam rubber) as it was in all 16 cases, the area of the scalp that supports the weight of the skull is damaged. The weight of the head over a number of hours damages the blood vessels of the scalp, either by direct traumatic crushing action, or more likely by blanching out the normally dense scalp vasculature and causing ischemia. The hair loss

is probably secondary to this vascular damage. Deep states of unconsciousness are required because the original injury would probably be quite painful to a conscious or sleeping patient.

The location of postoperative (pressure) alopecia is usually over the lower central vertex, because this is where the head rests when the neck is extended to keep a patent airway. It is generally not over the occiput where one would expect it if the person were in a normal recumbent position. However, case 7 had alopecia over the left occipital area because he was deliberately positioned to give the most exposure for right frontal craniotomy.

Of the 16 cases observed, 11 had anesthesia administered via endotracheal tube. With anesthesia of this sort, the anesthesiologist rarely touches or moves the head once the operation has started. This allows long steady pressure to be exerted on one area of the scalp. When anesthesia is administered via a mask, the anesthesiologist usually holds the mask and the patient's head. While doing this, he unintentionally moves the head, thus not allowing prolonged pressure to affect one scalp area. One factor that may be misleading in this is that many anesthesiologists prefer to use endotracheal anesthesia if they know that it will be a long operation. In none of the 16 cases did the patients have anything in their hair such as bobby pins, hair sets like braids, nor any other factor that might have caused local pressure.

Postoperative (pressure) alopecia is probably a preventable condition, although this has not been proven. A good preventive measure for any person who undergoes long anesthesia or who is in coma would be to have his skull supported by a doughnut-shaped foam rubber cushion or have his head repositioned periodically.

Postoperative (pressure) alopecia should always be ruled out when the diagnosis of alopecia areata is considered. Postoperative alopecia presents clinically as the rather sudden loss of hair over a circumscribed area of the posterior scalp. The alopecia is usually complete and the scalp otherwise appears normal. This appearance therefore is identical with that of alopecia areata except that alopecia areata sometimes has "exclamation point" hairs present. However, the natural history of the two diseases is different. Postoperative (pressure) alopecia is preceded by a period of unconsciousness, most commonly an operation, a number of days before the defluvium. The period of unconsciousness is often followed promptly by pain, swelling, or serous exudation and crusting in the area of the scalp upon which the head rested during operation. Hair loss then occurs in this site from 3 to 30 days postoperatively. The hair loss is temporary and regrowth always occurs within 120 days. The pathologic picture of postoperative (pressure) alopecia is quite different from that of alopecia areata. Alopecia areata differs in that it has no preceding period of deep unconsciousness, more than one spot may develop, regrowth does not always occur, and the alopecia may last more than 120 days before the hair reappears.

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