lungs in patients who have diffuse intravascular coagulation with fat embolism. This speculative connection between stress, hyperlipemia, and coagulation disturbances has stimulated investigators to look at levels and patterns of catecholamines, serum lipid fractions, and coagulation factors. Although the evidence is by no means consistent, it is anticipated that findings in these areas may provide more sensitive indicators of the fat embolism syndrome.[8, 9]

Conclusion

The diagnosis of fat embolism can be considered for any patient who develops tachycardia, fever, tachypnea, petechiae, or changes in sensorium early following fractures of a long bone. The diagnosis is supported by roentgenograms and decreasing hematocrit despite apparently adequate blood and fluid replacement. If these occur, arterial blood samples should be taken for analysis. The presence of hypoxemia without other explanation is diagnostic. Alterations in P_{CO_2} and *p*H are variable, but usually a compensatory respiratory alkalosis with an underlying bicarbonate deficit is found.

If morbidity and mortality are to be eliminated, careful observation of all patients for this disease is needed. Aggressive therapy should virtually eliminate mortality in the group at risk.

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CASE REPORTS

Problems Related to Aldosteronism during Cesarean Section

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Hypersecretion of aldosterone results in retention of sodium and urinary loss of potassium, with development of hypokalemic alkalosis. The clinical manifestations are hypertension, headache, paresthesias, intermittent tetany, periodic muscle weakness, polyuria and polydipsia. Primary aldosteronism (Conn's syndrome) is caused by an adenoma (adult form) or hyperplasia (juvenile form) of the adrenal cortex, while secondary aldosteronism is a complication of malignant and renovascular hypertension with excessive secretion of aldosterone due to stimulation by the renin–angiotensin mechanism. Primary aldosteronism rarely leads to peripheral edema or papil-

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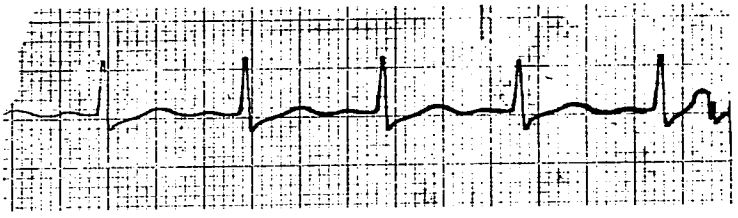

FIG. 1. Standard lead II of the electrocardiogram, taken after the patient had been placed on the obstetric operating table, showed marked depression of the S-T segment as well as decreased amplitude and broadening of the T-wave.

ledema, and renin production by the kidneys is usually suppressed. In contrast, papilledema is a constant finding in malignant and renovascular hypertension, and plasma renin activity tends to be elevated.[1-3]

The following case report describes a parturient woman in whom primary aldosteronism was diagnosed but not established definitely. However, the problems encountered during cesarean section were secondary to the electrolyte imbalance and not related to the type of aldosteronism.

REPORT OF A CASE

A 25-year-old para 2–0–1–0 was admitted by her obstetrician at 32 weeks' gestation with complaints of headache, muscle weakness and paresthesia (April 1969). A previous pregnancy, 1 year earlier, had been complicated by marked hypertension leading to a diagnosis of pre-eclampsia. However, following delivery of a premature stillborn, the blood pressure had remained elevated, and laboratory work-up had revealed increased excretion of potassium and aldosterone. The patient refused further tests and did not seek medical care again until she was in the second trimester of her second pregnancy. On admission to the antenatal

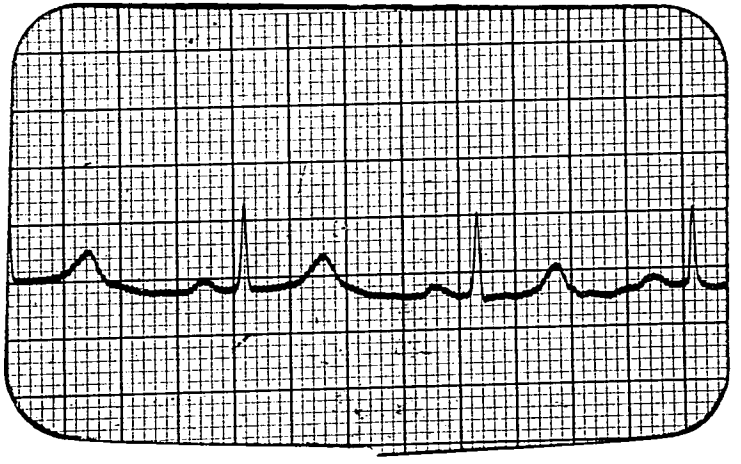

FIG. 2. Standard lead II of the electrocardiogram, taken after the patient had received approximately 900 mg of potassium chloride by rapid intravenous infusion, revealed disappearance of the abnormalities.

TABLE 1. Blood-gas and Acid–Base Findings at Time of Delivery

Blood	Maternal Artery	Umbilical Vein	Umbilical Artery
P_{O_2}	104	25	19
P_{CO_2}	37	45	49
pH	7.44	7.34	7.30
Base change	+1.0	−2.0	−3.0

unit, the blood pressure was 170/110 torr. Fundoscopy showed grade II retinopathy but no papilledema. A grade II/IV soft systolic murmur was audible. The uterus was enlarged in size to that of 28–30 weeks' gestation, with a fetal weight estimated at 1,000 to 1,200 g. No peripheral edema was demonstrable. Urinalysis revealed 3+ proteinuria. Serum electrolytes were: sodium 137 mEq/l, potassium 1.9 mEq/l, chloride 93 mEq/l, CO_2 content 32 mEq/l. The electrocardiogram was consistent with hypokalemia (S-T wave depression in leads V5 and V6 and a prominent U-wave) and left ventricular hypertrophy. A presumptive diagnosis of primary aldosteronism was made, and the patient was placed on bed rest and medicated with phenobarbital, methyldopa (Aldomet), spironolactone (Aldactone A), and potassium chloride elixir. On this regimen, the potassium rose to values ranging between 2.5 and 3.3 mEq/l. However, the blood pressure did not decline, and headaches became increasingly severe. Therefore, after four weeks of conservative treatment, on elective cesarean section was performed.

Preoperatively, the patient was given hydroxyzine (Vistaril), 25 mg, and atropine, 0.4 mg. She arrived in the obstetric operating room with a blood pressure of 190/100 torr. The electrocardiogram (limb leads) revealed regular sinus rhythm, but a markedly depressed ST segment and decreased amplitude and broadening of the T-wave (fig. 1). The attending cardiologist suggested rapid intravenous infusion of a potassium chloride solution (1,490 mg in 500 ml of 5 per cent dextrose in water). Following improvement of the amplitude of the T-wave (300 ml of solution) (fig. 2), anesthesia was induced with 225 mg of thiopental (Pentothal) and maintained with nitrous oxide–oxygen (6l:3l) via endotracheal tube and infusion of 0.1 per cent succinylcholine. The potassium chloride infusion was continued at a slower rate to completion. Nine minutes after induction (350 ml of potassium chloride solution), a 2,140-g male infant was delivered. He was impressively flaccid but had a good heart rate and made respiratory efforts (one-minute Apgar score 5, five-minute score 7). He required respiratory assistance (mask and oxygen) for six minutes. Umbilical cord blood-gas and acid–base data (table 1) did not offer an explanation for the extreme flaccidity. However, the potassium level in the infant's venous blood was high (table 2), and interference with neuromuscular function secondary to hyperkalemia was considered the most likely cause for the poor muscle tone. An electrocardiogram taken 30 minutes after birth showed no hyperkalemic characteristics and, therefore, no specific treatment was instituted. The potassium level declined gradually and reached normal values at 24 hours.

The mother's response to medical treatment improved postpartum, suggesting superimposed toxemia of pregnancy. Her potassium levels rose to between 3.2 and 4.3 mEq/, and her blood pressure declined to 150/90 torr. Following discharge, she again failed to return for further diagnostic work-up.

DISCUSSION

The combination of maternal hypokalemia and neonatal hyperkalemia has not been described before. To our knowledge, only two cases of primary aldosteronism in pregnancy have been reported.[2, 3] One of the patients was on spironolactone therapy but her infant was stillborn at 32 weeks' gestation; associated vaginal bleeding suggested abruptio placentae. The second patient received no specific treatment during a first pregnancy and delivered a normal child. During a second pregnancy, adrenal exploration revealed an adenoma of the left gland, which was removed, leading to complete recovery. The clinical course of our patient (absence of edema or papilledema and response to spironolactone and potassium chlo-

TABLE 2. Venous Blood Electrolytes of the Neonate during the First 48 Hours of Life

	Time after Birth					
	30 min	6 hours	8 hours	12 hours	24 hours	48 hours
Na^+	135	131	133	135	136	132
K^+	7.3	*	6.0	5.3	4.8	4.4
Cl^-	104	104	103	104	106	103

* Blood slightly hemolyzed.

ride) favors the diagnosis of primary aldosteronism. Confirmatory laboratory determinations (aldosterone secretion rate and plasma renin activity) were not done because their interpretation during pregnancy is difficult, since normal pregnancy is characterized by elevated aldosterone secretion and excretion as well as by elevated plasma renin activity.[3] Unfortunately, the adrenals were not explored at the time of abdominal delivery, and the patient did not submit to additional tests.

Adrenal function appears to be independent in mother and fetus. The presence of excess maternal adrenocortical hormone has been found to have little effect on the human fetus because the increased capacity of maternal plasma to bind steroid, relative to fetal blood, limits the passage of the hormone across the placenta.[4] The same fate may be expected to pertain to spironolactone, a 17-spirolactone steroid and a synthetic antagonistic analog of aldosterone.[5] Furthermore, spironolactone acts by blocking the sodium-retaining effects of aldosterone on the distal convoluted tubules, but in the fetus renal output is only a part of the large water turnover. Approximately 418 ml of water pass from the gastrointestinal tract, urine, lungs, and skin of the fetus to the amniotic fluid per hour, and 163 ml are absorbed by the fetus in the same period.[6] (The remainder of the water is removed via different pathways, *i.e.*, across the amnion on the fetal surface of the placenta, into the fetal vessels between chorion and amnion, and to the maternal extracellular space across the chorioamnion.)[7] Turnover of such amounts would tend to balance any effect of spironolactone, and the lack of spironolactone action in our neonate was confirmed by the normalcy of the serum sodium levels.

This, then, leaves the intravenously infused potassium chloride as the most probable cause of the neonatal hyperkalemia. There is no placental "barrier" to electrolytes, and drugs of low molecular weight diffuse freely across the placenta. With a molecular weight of only 74.54, potassium chloride must be expected to reach the fetus almost instantaneously. The effect of hyperkalemia in the infant differs little from that in the adult. "Symptoms are likely to be encountered when the serum potassium rises above 7 mEq/l."[8] The first manifestation is usually muscular weakness with loss of reflexes; at times a flaccid paralysis may develop; crying and breathing may be difficult. Derangements of cardiac function may occur, manifested by bradycardia or arrhythmia, and the electrocardiogram shows the typical picture of a tall pointed T-wave, a relatively low R-wave, and a wide QRS complex with prolonged Q-T interval.[8] At levels exceeding 8–9 mEq/l, the heart is rendered susceptible to vagal arrest.[9] We consider it fortuitous that only three-quarters of the potassium infusion had been administered at the time the infant was delivered, and we conclude that the intravenous administration of potassium salts to parturient women is hazardous to the fetus. Therefore, the benefit to the mother ought to be weighed carefully against the potential danger to the fetus and neonate.

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