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Anhydramnios

Anhydramnios is the complete or nearly complete absence of measurable amniotic fluid surrounding the fetus. It must be distinguished from oligohydramnios, in which fluid is reduced but still detectable. During early gestation, amniotic fluid is derived partly from maternal and placental sources, but fetal urine becomes its principal component during the second trimester. Normal volume also depends on fetal swallowing, pulmonary fluid secretion, membrane integrity, and fluid movement across the placenta and fetal membranes. Consequently, anhydramnios is not a diagnosis by itself but a manifestation of an underlying fetal, placental, or membrane disorder.

The timing, cause, and duration of anhydramnios largely determine its consequences. Early pregnancy renal anhydramnios is generally defined as absent fluid caused by fetal renal failure by approximately 22 weeks of gestation. Its principal causes are bilateral renal agenesis, severe cystic or dysplastic kidney disease, and lower urinary tract obstruction accompanied by irreversible renal damage. Other causes of absent fluid include preterm prelabor rupture of membranes and severe uteroplacental insufficiency. These conditions have different prognoses and therapeutic options; therefore, accurately identifying the etiology is essential before counseling the family or considering fetal intervention.

Amniotic fluid is critical for pulmonary development. The fetal lungs normally secrete fluid into the tracheobronchial tree, creating internal pressure that promotes airway branching and lung growth. In the absence of surrounding amniotic fluid, lung fluid can escape more readily through the fetal airway during glottic opening. Loss of intrapulmonary pressure, restriction of fetal breathing movements, and compression of the fetal thorax interfere with normal lung expansion. Persistent anhydramnios can consequently produce pulmonary hypoplasia, pulmonary vascular abnormalities, severe neonatal respiratory failure, and persistent pulmonary hypertension. The greatest vulnerability appears to occur during the canalicular phase of lung development, although clinically important pulmonary injury can also develop when anhydramnios begins after 22 weeks.

The traditional view that fluid-dependent lung development is essentially complete by 22 weeks is not supported by the available clinical evidence. Persistent renal anhydramnios beginning later in the second trimester can still cause lethal pulmonary hypoplasia. Fetuses with previously normal fluid who develop anhydramnios between approximately 22 and 30 weeks may experience profound respiratory morbidity if the fluid deficit remains uncorrected. Restoration of amniotic fluid or successful relief of urinary obstruction may permit survival in selected cases. Gestational age at onset should therefore inform prognosis, but it should not be used as an absolute boundary beyond which anhydramnios

is considered harmless or fetal treatment automatically futile.

Prenatal ultrasound is the initial diagnostic examination. Evaluation should document whether any measurable pocket of fluid remains and should assess the kidneys, renal arteries, ureters, bladder, urethra, external genitalia, and the remainder of the fetal anatomy. Bilateral renal agenesis is suggested by absent kidneys and renal arteries, failure to visualize the bladder, and progressive loss of fluid. Dysplastic kidneys may appear enlarged, echogenic, cystic, or severely involuted. Lower urinary tract obstruction is characterized by an enlarged and thick-walled bladder, possible dilation of the ureters and renal collecting systems, and variable renal echogenicity or cystic dysplasia. A small or nonfilling bladder in an obstructed fetus may indicate advanced loss of renal function.

The evaluation must also exclude ruptured membranes, particularly when renal anatomy does not clearly explain the absent fluid. Maternal history, sterile speculum examination, and biochemical testing of vaginal fluid may be required. A detailed anatomic examination is important because associated malformations may indicate a syndromic condition, change the expected outcome, or preclude intensive neonatal treatment. Severe anhydramnios can limit sonographic visualization; a diagnostic amnioinfusion may temporarily restore an acoustic window, confirm membrane integrity, improve assessment of fetal anatomy, and clarify the presence of renal tissue, renal blood flow, or additional abnormalities. Fetal magnetic resonance imaging can provide supplementary information about lung volume and anatomy when ultrasound remains inadequate.

Genetic assessment is an important component of the evaluation because renal agenesis, cystic renal disease, dysplasia, and obstructive uropathy may be associated with chromosomal abnormalities or single-gene disorders. Testing may include karyotyping, chromosomal microarray analysis, targeted testing, multigene panels, or exome sequencing, depending on the phenotype and family history. A nondiagnostic genetic result does not exclude a genetic disorder, while identification of a pathogenic abnormality can substantially alter counseling regarding recurrence, associated disease, neonatal treatment, and the appropriateness of fetal intervention.

Management should begin with multidisciplinary, nondirective counseling. Options include termination of pregnancy when legally and clinically available, expectant management with perinatal palliative care, and carefully selected fetal treatment. Counseling must separate pulmonary prognosis from renal prognosis. Restoring amniotic fluid may enable lung development, but it does not create functioning kidneys or reverse established renal dysplasia. A newborn who survives respiratory transition may still have end-stage kidney disease requiring immediate and continuous renal replacement therapy. The relevant outcome is therefore not simply live birth or early respiratory survival, but the possibility of prolonged hospitalization, dialysis, serious complications, kidney transplantation, and an acceptable long-term quality of life.

Serial amnioinfusion is intended to reduce pulmonary hypoplasia by repeatedly replacing the absent fluid with warmed isotonic solution under ultrasound guidance. Treatment

generally begins before 26 weeks and is repeated according to fluid loss and gestational age, often at intervals of several days to approximately one week. The objective is to maintain a fluid volume compatible with continued lung growth until delivery. In carefully selected fetuses with isolated bilateral renal agenesis, serial amnioinfusions have clearly demonstrated that lethal pulmonary hypoplasia is not inevitable. Among 17 live-born infants treated prenatally, 14, or 82%, survived for at least 14 days and underwent placement of dialysis access.

Short-term respiratory success, however, did not translate into comparable long-term survival. Only six of the 17 live-born infants, or 35%, survived to hospital discharge while receiving chronic peritoneal dialysis. Subsequent deaths occurred from infectious and cardiopulmonary complications. Neurologic injury, including stroke, emerged as a particularly concerning complication among infants receiving dialysis. Hypotension, infection, nutritional failure, prematurity, cardiovascular instability, and technical difficulties with dialysis access also contributed to morbidity. At the time these outcomes were reported, none of the surviving children had yet undergone kidney transplantation. Transplantation generally requires sufficient somatic growth, and the absence or severe abnormality of the lower urinary tract can complicate urinary reconstruction.

Prenatal treatment also carries obstetric risks. Preterm prelabor rupture of membranes occurred in approximately 61% of treated pregnancies, chorioamniotic separation in 28%, and vaginal bleeding in 22%. All treated pregnancies delivered prematurely, with a median gestational age of approximately 32 weeks. Greater gestational age and higher birth weight were associated with better neonatal survival, while premature membrane rupture reduced the interval available for treatment and lung growth. Serious maternal injury was uncommon, but repeated invasive procedures may cause discomfort, hospitalization, infection, bleeding, membrane complications, fetal loss, and spontaneous preterm delivery. Maternal safety and neonatal benefit must therefore be reassessed throughout treatment.

Lower urinary tract obstruction represents a different therapeutic situation because relieving the obstruction may restore fetal urine production when renal function remains sufficiently preserved. Vesicoamniotic shunting and fetal cystoscopic treatment can decompress the bladder and potentially replenish amniotic fluid naturally. These interventions may improve perinatal survival, but they do not reliably preserve long-term renal function. Vesicoamniotic shunts may dislodge, migrate, retract, or become obstructed and can cause fetal ascites, abdominal-wall injury, membrane rupture, preterm labor, or fetal death. Available evidence suggests improved survival during the perinatal period but no established improvement in survival beyond infancy or in renal function at two years.

Selection for intervention in lower urinary tract obstruction should therefore consider gestational age, bladder appearance, renal echogenicity, cystic dysplasia, urinary biochemistry when obtainable, associated anomalies, and evidence of residual urine production. A severely damaged urinary system may not recover after decompression. Conversely, successful correction of an obstruction with resolution of anhydramnios may reduce pulmonary injury and improve the opportunity for neonatal treatment. When

obstruction cannot be corrected or renal failure persists, serial amnioinfusion may theoretically support lung development, but the child will still face the consequences of advanced kidney disease.

Postnatal care requires immediate access to advanced respiratory, nephrology, surgical, and intensive-care support. Lung-protective ventilation, high-frequency ventilation, inhaled nitric oxide, cardiovascular support, and treatment of pulmonary hypertension may be necessary. Extracorporeal support cannot compensate for an extreme absence of functional lung tissue and may be inappropriate when recovery is not physiologically possible. If respiratory stabilization succeeds, peritoneal dialysis is generally the preferred long-term renal replacement strategy, although very low birth weight, abdominal complications, infection, hypotension, and lack of usable urinary anatomy can make treatment difficult.

Anhydramnios should ultimately be understood as a time-sensitive, etiologically diverse condition rather than a uniformly lethal diagnosis. Prenatal fluid restoration can mitigate pulmonary hypoplasia and permit short-term survival in selected fetuses, but it transfers the principal threat from respiratory failure to severe chronic renal disease and its complications. Current evidence supports comprehensive diagnostic evaluation, genetic testing, transparent counseling, and individualized decision-making. Serial amnioinfusion for renal agenesis or irreversible fetal renal failure should remain restricted to highly specialized, ethically supervised treatment protocols with mandatory long-term follow-up. Meaningful counseling must present both the possibility of pulmonary rescue and the substantial probability of prematurity, dialysis-dependent survival, neurologic injury, prolonged hospitalization, and death before transplantation.

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Subcutaneous Emphysema

Subcutaneous emphysema in children is one of those findings that looks far more alarming at the bedside than it usually turns out to be. A child arrives with a puffy, swollen neck or chest, and when a clinician presses a finger against the skin, it crackles under the touch — a sensation clinicians often describe as walking on fresh snow. That crackling, called crepitus, comes from air that has escaped its normal home inside the airway and tracked its way into the soft tissue layers beneath the skin. It is a striking physical sign, and it tends to trigger a cascade of imaging and specialist consultations, but in the great majority of pediatric cases the underlying process is benign and self-limited. Understanding why air ends up where it shouldn't be, and what conditions tend to produce it in children, helps explain why the same physical finding can mean anything from a mild, spontaneously resolving nuisance to a marker of a genuine surgical emergency.

The basic physiology is fairly consistent across nearly every cause. Somewhere in the respiratory tract — usually at the level of the small alveoli, sometimes higher up in the trachea or major bronchi — a tear or rupture allows air to escape from where it belongs into surrounding tissue planes. Once air is loose in the chest, it tends to follow the path of least resistance, tracking along the sheaths that surround blood vessels and airways toward the central portion of the chest, the mediastinum. From there it can continue migrating upward into the neck and face, or outward into the chest wall, spreading through fascial planes the way water might spread through loose soil. This central escape of air into the mediastinum is called pneumomediastinum, and it very often travels hand in hand with subcutaneous emphysema, since the same forces that push air into the mediastinum also push it outward under the skin.

In children, the leading cause of this whole cascade, when there's no obvious traumatic explanation, is asthma. A child having a significant asthma flare is fighting to move air past narrowed, inflamed airways, and that fight can generate surprising pressure swings inside the chest. Violent coughing, forceful exhalation against a nearly closed airway, or the kind of breath-holding and straining that happens during a bad wheeze can all raise the pressure inside the smallest air sacs enough to rupture their delicate walls. Air that escapes this way doesn't usually cause immediate danger — it isn't like a burst balloon so much as a slow leak — but it dissects along the same vascular and airway sheaths, eventually surfacing in the neck, the collarbone area, or across the upper chest, sometimes only becoming apparent to a worried parent who notices swelling or an odd crunching feeling when they touch their child's neck. The classic story is a child, often in the older end of childhood or adolescence, who already carries an asthma diagnosis and turns up with chest pain, breathlessness, and that telltale puffiness after a flare-up. Interestingly, this complication of asthma can also be the very first sign that a child has asthma at all, appearing during someone's first-ever wheezing episode before any formal diagnosis has been made. Boys and girls appear to be affected in roughly equal numbers, and while it can happen at almost any pediatric age, adolescents and children with a history of poorly controlled disease seem to be at greatest risk. Reassuringly, when doctors have tracked outcomes across many such cases, nearly everyone recovers fully with nothing more than supportive care: rest, oxygen if needed, pain control, and treatment aimed at getting the

underlying asthma back under control. The subcutaneous air and the pneumomediastinum tend to resolve on their own over several days to about a week, and only rarely does a child develop something more worrying, such as a collapsed lung, that would call for more invasive intervention like a chest drain.

Beyond asthma, there is an entirely separate category of cases where the trigger is procedural rather than a disease flare. Children who undergo tonsil and adenoid removal, one of the most routine operations in pediatric surgery, occasionally develop subcutaneous emphysema afterward, sometimes dramatically so. The working theory in these cases involves the deeper layers of tissue at the back of the throat, just beneath where the surgical instruments operate. If those layers are breached — sometimes helped along by a mouth-holding device used to keep the jaws open during the procedure, sometimes by the mechanics of the child coughing, swallowing, or straining while waking up from anesthesia with a tube still in place — air can be forced into the space behind the throat and travel from there down into the chest and out into the surrounding soft tissue. In some documented cases, a small tear was later found not in the surgical field itself but lower down, in the windpipe, raising the possibility that the injury actually came from the breathing tube or the awakening process rather than from the operation. These post-surgical cases can look dramatic, with swelling that spreads from the face and neck down across the shoulders and chest, but the pattern of recovery tends to mirror the asthma-related cases: supportive monitoring, sometimes a chest tube if there's an associated collapsed lung, and steady resolution over several days.

A third scenario, and one that carries real diagnostic urgency, is the possibility of an inhaled foreign object sitting somewhere in the windpipe or bronchi. Young toddlers explore the world with their mouths, and small objects — seeds, nuts, bits of food, small toy parts — can slip past the vocal cords and lodge in the airway. Sometimes this produces the dramatic, unmistakable choking episode that sends a parent racing to the emergency department. But sometimes it doesn't. A foreign body can sit quietly for days or even weeks, causing only a nagging cough, before it starts acting like a one-way valve: air gets past it on the way in but can't fully escape on the way out, causing pressure to build up behind it until something gives, and air leaks out into the surrounding tissue. In children who present with unexplained subcutaneous emphysema and no obvious asthma or trauma history, especially toddlers, this possibility has to stay near the top of the list, because the definitive treatment — locating the object and removing it through a scope passed into the airway — is completely different from watchful waiting.

Yet another pathway runs through the operating room itself, specifically through the act of placing a breathing tube. Even when intubation goes smoothly and is performed by an experienced team, the tissues of a small child's airway are delicate, and on rare occasions the tube itself, or the pressure of mechanical ventilation, causes a tiny injury that lets air escape. What makes these cases especially tricky is that the swelling doesn't always show up right away. A child can seem completely fine after anesthesia, only to develop puffiness and crackling under the skin hours later, sometimes triggered by something as simple as

crying, which raises pressure in the chest the same way coughing does. When this happens, doctors typically move quickly to imaging to look for an associated collapsed lung, since that combination can worsen quickly and may need drainage, even though the underlying tissue injury itself usually heals on its own once the pressure is relieved.

Finally, there remains a smaller group of children in whom no cause is ever found at all — no asthma, no surgery, no foreign body, no difficult intubation, sometimes not even a preceding cough or fever severe enough to explain it. This idiopathic pattern, sometimes given its own name in the medical literature, seems to follow the same basic mechanism of alveolar rupture and air migration, just without an identifiable trigger. It has been described even in toddlers barely over a year old, presenting with fever, cough, and breathing difficulty, and after every more dangerous explanation has been ruled out through careful examination and imaging, these children too tend to do well with nothing more than close observation.

Across all of these very different starting points, the underlying story is strikingly consistent: air escapes where it shouldn't, travels along predictable anatomical pathways, and in the overwhelming majority of children resolves quietly with time, oxygen, rest, and treatment of whatever set the process in motion in the first place. The real clinical work lies not in treating the air itself, which the body handles well on its own, but in figuring out, case by case, what let it escape.

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Artery of Adamkiewicz

The spinal cord depends on a surprisingly fragile network of blood vessels, and among these, one artery stands out for the outsized role it plays in keeping the lower spinal cord alive. Known as the artery of Adamkiewicz, or by its older name arteria radicularis magna, this vessel is the single largest contributor of blood to the anterior spinal artery along the thoracolumbar region. While the spinal cord as a whole receives blood from multiple small radicular branches coming off the aorta, most of these vessels regress during development,

leaving only a handful of dominant feeders. In the vast majority of people, one vessel clearly outranks the others in size and importance, and it is this vessel that carries the name of the nineteenth-century physician who first described it in detail.

Anatomically, the artery arises from an intercostal or lumbar branch of the descending aorta, most often on the left side, and enters the spinal canal through an intervertebral foramen alongside an exiting spinal nerve root. Once inside the canal, it turns sharply upward, forming what is often described as a hairpin or "candy-cane" curve, before merging with the anterior spinal artery. This anterior spinal artery, in turn, supplies roughly the front two-thirds of the spinal cord, an area responsible for motor function and for the pathways carrying pain and temperature sensation. Because the artery of Adamkiewicz is so often the dominant supply to this region in the lower thoracic and lumbar cord, any interruption of its flow can produce a strikingly consistent pattern of injury: sudden weakness or paralysis below the level of damage, along with loss of bladder and bowel control, while the sense of touch and joint position often remains intact, since those pathways travel through a different part of the cord that receives blood from other vessels.

What makes this artery clinically important is not just its role in normal physiology but its extraordinary variability from person to person. Its point of origin can be as high as the mid-thoracic spine or as low as the upper lumbar region, and while it favors the left side in the great majority of individuals, a meaningful minority have it entering from the right, or in rare cases have more than one such vessel altogether. This unpredictability means that a surgeon cannot simply assume where the artery will be based on textbook averages. A vessel that supplies such a large portion of the spinal cord, yet whose location differs so much between patients, becomes a genuine hazard whenever an operation approaches the aorta, the vertebral column, or the tissues surrounding the spinal canal.

In adults, this concern arises most often during surgery on the thoracic or abdominal aorta, where aneurysm repair or stent placement can interrupt segmental arteries feeding the spinal cord and cause devastating paralysis if the artery of Adamkiewicz is sacrificed without warning. But the same anatomical hazard extends squarely into pediatric surgery, and in some ways it becomes even more consequential in children because of the types of tumors that develop close to the spine early in life. Posterior thoracic and paraspinal masses, most commonly neuroblastic tumors such as neuroblastoma, ganglioneuroblastoma, and ganglioneuroma, frequently arise directly beside or even within the neural foramina through which this artery passes. Some of these tumors grow in a distinctive dumbbell shape, extending from the chest cavity into the spinal canal through the very foramen the artery uses to reach the cord. Operating to remove such a tumor completely, without leaving behind malignant tissue that could recur, means working in immediate proximity to a vessel whose injury could leave a child permanently paralyzed or incontinent.

This is precisely why identifying the artery before surgery has become such a central concern in the care of children with these tumors. Surgeons and radiologists have several imaging tools available, including computed tomography angiography and magnetic

resonance angiography, both of which are noninvasive and can often reveal the vessel's course without exposing a child to the risks of a catheter-based procedure. Yet for decades, the recognized gold standard for visualizing spinal blood supply has been catheter spinal angiography, a more invasive test in which contrast is injected directly into the small arteries feeding the spinal cord. Because it directly opacifies the vessel rather than relying on indirect signals, angiography tends to succeed in locating the artery more consistently than cross-sectional imaging alone, particularly in small children where vessel caliber is tiny and imaging resolution is more limited. Encouragingly, when this invasive test has been used in children, it has proven remarkably safe, with essentially no reported complications directly attributable to the procedure itself, even in infants weighing only a few kilograms.

The payoff for this extra effort appears to be real. When surgeons know beforehand exactly where the artery enters the spinal canal, they can plan their operative approach, choose which side to work from, decide how aggressively to dissect near the foramen, and adjust their technique to avoid the vessel entirely, or deliberately preserve it if the tumor surrounds it. Children who underwent successful preoperative identification of the artery experienced markedly fewer neurological complications after surgery compared to those in whom the vessel's location remained unknown, and in some reported groups, no child with a properly identified artery went on to develop paralysis or other neurologic sequelae afterward. This does not mean imaging guarantees complete removal of every tumor, since some masses so thoroughly encase the artery that surgeons choose to leave a small remnant of tumor behind rather than risk the child's mobility and continence, a trade-off that speaks to how seriously this single blood vessel is regarded in the operating room.

Beyond the tumor setting, the same anatomical hazard occasionally intersects with other procedures performed near the spine, including epidural injections, vertebral fracture treatments, and instrumented spinal fusion, all of which carry a small but recognized risk of catastrophic cord injury if a needle, screw, or cement inadvertently damages this vessel or its branches. Across every context in which it appears, the lesson remains the same: a structure barely a millimeter wide, tucked into an unpredictable location along the spine, can determine whether a patient walks away from surgery or spends the rest of their life dealing with paralysis, making careful preoperative attention to the artery of Adamkiewicz one of the most important anatomical considerations in modern pediatric surgical oncology and spine care.

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