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Housestaff Puzzler “Unilateral Arm Swelling”

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HPI: SH is a 17 year old African-American female who presented to the BAMC Adolescent clinic with a 3 week history of right upper arm swelling. The swelling was initially noted after a track and field event and was seen in the Emergency room the following day, diagnosed with a muscle strain and sent home with ibuprofen. The swelling had actually improved over the last week, however, her father brought her into the clinic on the day of presentation because he noticed her right upper arm veins were “bulging.” She reported a constant, dull pain that was worse with movement, but was also mildly improved since the symptoms first presented. SH denied any trauma to the area, but reported that she was a relay track runner and uses the right arm to receive the relay baton from her teammates. She denied any loss sensation, weakness, numbness or tingling in her right arm. She denied any

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Meconium Stained Amniotic Fluid (MSAF) in the Delivery Room

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There are few common occurrences in newborn medicine as anxiety provoking as the delivery of an infant through meconium stained amniotic fluid (about 10% of all deliveries). Worse yet, the seemingly ever-shifting recommendations and the conflicting messages in the NRP (2000) guidelines add to the anxiety about delivery room management of infants who can develop a life threatening condition (meconium aspiration syndrome, MAS) that for several decades was considered preventable with appropriate delivery room management. Imagine the delivery of a term infant born through meconium. Does it matter whether it’s thin or thick? Does it matter *when* OB suctions the mouth and nose, and what they use (deLee or bulb)? Do you prevent spontaneous respiration by squeezing the chest or putting a thumb in the baby’s mouth on the way to the warmer? How do you decide which baby gets immediate intubation? What’s the endpoint for intubation and suction; some finite number of times or until there’s no more meconium returned? When do you abort suctioning and move into resuscitation? Worst of all, if the infant ends up with MAS, was it because you didn’t suction well enough (if at all)? Think about the first question asked during the presentation of a baby who develops MAS: “was the baby suctioned in the DR?”

Since before time itself we’ve known that meconium at delivery is associated with

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New Changes in Pap Smear Recommendations

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Part of preventive pediatric health care for adolescent females involves guidance on Sexually Transmitted Infection (STI) risk behavior, screening, treatment, and prevention. Included in this topic is routine Pap smear screening, and understanding when referrals are necessary for this care. The AAP among other organizations has published recommendations concerning Pap smear screening. With new information and understanding about abnormal pap results, the American Cancer Society has updated these guidelines with some

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worse outcome (infection, need for resuscitation, MAS, death). Getting meconium out of the lungs has been a goal of newborn medicine for more than the 30 years that it's been subjected to scientific "study". The modern (post "medline" entry) era of management started with a series of papers from Gooding and Gregory, Ting and Carson, which are considered "landmark" studies. In an attempt to produce an animal model in which to study the benefits of steroids on the treatment of MAS, Gooding and Gregory discovered that they couldn't induce an MAS type CXR in a helpless newborn puppy unless they instilled a meconium slurry directly into the trachea before the first breath. This led them to conclude that meconium in the trachea was a necessary precursor to MAS. The three landmark studies concluded several things. 1) Suctioning meconium from the trachea before the first breath decreased mortality, 2) absence of meconium in the hypopharynx did not mean that the trachea was clear, 3) infants who developed respiratory symptoms after birth were less likely to have been suctioned at birth and 4) combined OB suctioning and "indicated" pediatric suctioning (if there was mec visible at the cords) decreased morbidity over pediatric suctioning alone or OB suctioning and pediatric suctioning with saline lavage in the delivery room. Apparently the 1970s was not an era encumbered by the need to prove things in a controlled way or to use statistics, and these studies all have serious methodological flaws that in today's evidence based approach to medical literature would have been considered "fatal." Nevertheless, they show how poor data can be used to support a conclusion if it's strongly worded enough ("we do not believe that a controlled study could ever be done because of the

high mortality rate associated with meconium aspiration pneumonitis", in other words: do what we say or you can be accused of actively killing babies through negligence). But that's a separate story.

Since that time, there have been a number of studies in both humans and animals that have guided practice to its current form, which is

- 1) Meconium is bad, very very bad
- 2) It doesn't matter if meconium is thin or thick when an infant is depressed
- 3) Infants in MSAF benefit from amnioinfusion.
- 4) Infants who are born through meconium get OP suctioning by OB on the perineum before the chest is delivered.
- 5) Depressed infants get immediate tracheal suctioning (until clear)
 - a. unless the infant requires further resuscitation
- 6) Active kids (HR>100, good tone and respiratory effort) get no benefit from empiric tracheal suctioning at birth
- 7) MAS can happen with gasping in-utero, but some happens after delivery and appropriate DR management has been shown to decrease the incidence of MAS.
- 8) Not all respiratory distress born through meconium is MAS

Taking each one of these in turn we'll review the evidence behind the statements.

- 1) Meconium is bad, very very bad

This is an empiric statement. Comparisons are made between MASF babies and non-MASF babies and the MASF babies always have worse outcomes. They also are older, as a group. The significance is that advanced age leads placental insufficiency in post-mature infants. Beyond 42 weeks gestation, both morbidity and mortality increase.

Prior to the early 80s, stopping gestation at 42 weeks was not a standard and the group of infants born through meconium often included kids up to 46 weeks gestation.

Meconium is excreted as part of the normal maturation process. But a good hypoxic event leads to meconium excretion as well (through the sympathetic nervous system, think about "scaring the 'mec' out of someone"). We'll touch on that in the next section. So MASF can be a marker of fetal maturity but it can also be a marker of significant in-utero stress. And in-utero stress is bad. For infants born at <37 weeks gestation, in fact, meconium is almost always a sign of in-utero distress. About 28 weeks is as young as meconium excreters can get (based on personal observation). The biggest difficulty people have when talking about meconium is the feeling that meconium is somehow the cause of this increased morbidity, and not just the marker. It certainly complicates an aspiration syndrome, but it doesn't cause one.

- 2) It doesn't matter if meconium is thin or thick when an infant is depressed
- 3) Infants benefit from amnioinfusion

In the old days it seemed to matter, but NRP now says it doesn't. If you have meconium in the face of being totally healthy and it's just a sign of maturity then it will be a little meconium in a lot of amniotic fluid, thus "thin". But if you are a stressed baby with placental insufficiency you will put out more meconium (stress) in less fluid (oligohydramnios) leading to "thick" meconium. The problem now is that with amnioinfusion you can thin out meconium without too much difficulty. However, just taking away the meconium doesn't necessarily take away the stress that led to the meconium excretion. The amnioinfusion may relieve an

umbilical cord compression (one hypothesis), but either way, amnioinfusions do reduce the risk of MAS and mec below the cords in infants with thick MSAF, distress and oligohydramnios.

If you take all infants born through meconium and compare them to infants not born through meconium, the infants born through meconium have a higher incidence of problems. However, if you separate out “thin” and “thick” meconium, “thin” meconium does not appear to be associated with an increased incidence of problems, but “thick” does. But in the absence of any supporting data *specifically* in depressed kids, the folks at NRP have decided to recommend treating all meconium as meconium, no matter how thick or thin.

4) Infants who are born through meconium benefit from OP suctioning by OB on the perineum (or surgical field) before the chest is delivered.

This is the overriding hypothesis of the Carson study, and the big message that comes from it. It’s based on the work of a gentleman named John Johnson, who stuffed esophageal manometers into babies after delivery of the head and before delivery of the chest (apparently in the age before informed consent). What he found was that the recoil of the ribcage after emergence from the vaginal canal led to about a 40cm H₂O pressure drop in a sort of gasp. Carson’s contemporaries felt this “gasp” was when meconium could move from the oropharynx to the trachea, and thus lead to MAS. So suctioning out the OP before the chest delivers is the rational way to manage kids born through MAS. Therefore, once the baby is delivered, the chest is already recoiled and there should be no benefit to OP suctioning. If it wasn’t done while the chest was in the canal, it’s too late. This is the origin of the now-

discarded idea of squeezing the chest after delivery to prevent further aspiration.

But the story doesn’t end there, because you can suction with deLee and you can suction with a bulb. Which is better? It turns out that unless you’re a cat, it doesn’t matter. Either method is associated with the same incidence of meconium below the cords (about 30% post 1980s) and MAS (about 2% of those born through MSAF). The bigger question is whether OB suctioning on the perineum actually impacts mec below the cords and MAS. Non-randomized series say that it doesn’t, and it’s never been subjected to a randomized controlled trial. It’s a good sounding idea that became a standard of care in the 70s.

So, if OB is going to suction, it doesn’t matter how they do it but they need to do it before the chest is delivered.

5) Depressed infants get immediate tracheal suctioning (until clear)

a. unless the infant requires further resuscitation

This is the heart of the new recommendations from the NRP. It was a universal recommendation for all infants born through thick meconium until the vigorous kids fell out (see next section). The ILCOR advisory statement, on which NRP is based, says that if a kid is depressed, as long as the HR is >60 you keep suctioning until the airway no longer produces meconium on suction. If the HR drops below 60, resuscitate per the routine without meconium. The process of drying and stimulating is to be delayed but purposely depressing the infant (thumb in mouth, chest squeeze) is no longer recommended.

6) Active kids (HR>100, good tone and respiratory effort) get no benefit from empiric tracheal suctioning at birth

This recommendation freaked

out a lot of pediatricians. All the sudden, the one thing you could do to help infants born through meconium, that one skill that separated a pediatrician from a baby-technician, was no longer recommended. The fear was that you’d now allow some kids to develop MAS just because not all kids *needed* suctioning. To this point, which camp you fell in to depended on who you read. “Intubators” followed the Gooding/Gregory, Ting, Carson and earlier Wiswell line of reasoning that intervention was *clearly* needed to improve outcome. This was not a lonely camp. But as soon as the idea was proposed, the literature was full of doubters who showed that although the level of intervention increased, the outcomes remained the same. The strongest arguments for universal intubation up until Dr. Wiswell’s massive multi-center RCT, published in 2000 were made, in fact, by Dr. Wiswell. Starting with a retrospective review of the military database of births he showed that by logistic regression there was a significant steady decline in the incidence of MAS since the introduction of OB and pediatric intervention. Familiarity with the use and abuse of logistic regression is important. While the line clearly was not flat (thus significant, $p < 0.05$), the R² value was about 0.25. This means that the actual change in data accounted for about 25% of the slope of the regression line. Hardly a convincing correlation. In review, it’s likely that a good deal of change in the numbers (from 8% down to a low of about 4%, still high relative to most literature) were probably related to a gradual appreciation that not all respiratory distress born through meconium was MAS, and a decrease in the high-risk post-dates deliveries. Of interest is the concept that if you are interrupting the pathogenesis of MAS so cleanly, why is there only a gradual change in the incidence of MAS over time, and not

a step-off. Also consider how much meconium you get during a typical suctioning. Usually a few milliliters at best, rarely at most 7-10ml of meconium tinted fetal lung fluid. In the face of a 60-200 ml functional residual capacity of the lung after birth for a 3-4kg infant does not seem like it should cause that much problem. Almost all the animal models of MAS have the shortcoming of instilling a meconium slurry into already transitioned lungs and are thus a good model of meconium aspiration pneumonitis. The ball-valve effect of the particles on the airways leads to air trapping (with or without pneumothorax) with patchy areas of atelectasis and over inflation that are typically seen with what's diagnosed as MAS. Inactivation of surfactant and an inflammatory response are also seen. However, Meconium Aspiration Syndrome is *more* than just an aspiration. The highest incidence is in kids born through meconium who are SGA or IUGR and post dates with evidence of placental insufficiency. They are chronically stressed in utero and develop the makings of PPHN (see section of PPHN) before they're even born. Gasping in utero leads to recruitment of patchy areas of alveoli filled with whatever is in the amniotic fluid. The chronic in-utero hypoxia leads to stress that leads to meconium excretion and leads to remodeling of the pulmonary vasculature. Because it's easier for hypoxia to lead to excretion of meconium than cause PPHN, you are more likely to see term kids with PPHN who have meconium than not.

The difference between depressed and active kids lies in what happens to the flow of fetal lung fluid. During well being, fetal lung fluid has a net flow out of the lung, but during asphyxia there is gasping of amniotic fluid and contents into the lung. At least in the lamb, if asphyxia is reversed, the net outward flow starts up again and

aspirated meconium is cleared from the trachea. So in the depressed kids who may not have cleared out their tracheas, suctioning still has some basis in rationality. Although depressed infants haven't been studied separately, remember that being active at birth (roughly 75%) is still associated with about a 2-3% incidence of MAS in those born through MSAF, regardless of intubation intent. Also remember the big caveat of the Wiswell study: just because you don't intend to intubate, significant respiratory distress or evidence of obstruction (~6% occurrence) should still lead to intubation and tracheal suctioning, and about 20% of these kids go on to develop MAS.

7) MAS can happen with gasping in-utero, but some happens after delivery and appropriate DR management has been shown to decrease the incidence of MAS.

Along with their early study, Gooding and Gregory got CXRs on all infants born through meconium. In a blinded fashion, 33% of the infants who were asymptomatic were found to have abnormal CXR (12% were "moderately" to "severely" abnormal). That worked out to a 30% positive predictive value for an abnormal CXR to predict symptoms. To predict MAS obviously lower. A 1995 paper by Davis looked at a series of 12 kids who *died* of MAS, 8 of which were autopsied. Of those 8, only 4 had meconium in their lungs, 2 others had evidence of amniotic fluid aspiration but no meconium and 2 didn't have any evidence of aspiration. In the two kids with aspiration but no meconium, they must have aspirated before excretion of meconium.

Autopsy of stillbirths can reveal evidence of aspirated meconium even without ever having had a spontaneous breath, so we know MAS can happen in utero. But despite diligent effort to intervene in the DR after birth of the baby, there

are no changes in the incidence of MAS (or of meconium below the cords). This brings into question whether MAS ever happens during or after delivery. Anecdotal experience still suggests it might.

8) Not all respiratory distress born through meconium is MAS

Meconium complicates an aspiration syndrome, but aspiration syndrome can happen in the absence of meconium. There's still TTN, RDS in the diabetic infants, PPHN, pneumonia, pneumothoraces etc. that happen both with and without meconium present.

So, in summary in the delivery room as the evidence now stands:

- Amnioinfusion works for kids with evidence of compromise
- OB suctioning on the perineum or at the incision site should be done before delivery of the thorax.
 - Bulb or deLee are equally effective
 - Kids who have good tone, respiratory effort and HR > 100 should be managed expectantly (75% of kids born through MSAF)
 - Still be prepared to intubate and/or clear the OP with signs of obstruction or distress (~6% of those treated "expectantly")
 - Kids who are less active than above (poor tone, weak resp effort) should be quickly intubated and suctioned
 - NRP recommends until clear or until HR < 60
 - For practical purposes, probably not more than 2 quick passes, or one slow pass
 - MAS *may* be partially preventable and tracheal suctioning may reduce the severity of illness but this is not an evidence-based statement.
 - MAS does happen before delivery and therefore cannot be entirely prevented.
 - So don't feel guilty about

not suctioning enough, there's no evidence the practice has any impact on ultimate outcome.

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bruising or discoloration to her hand or arm. She denied any shortness of breath, dyspnea, fever, chills, night sweats or weight loss.

Physical exam: the patient was a thin, well-developed, athletic female in no acute distress. Heart rate 65, blood pressure 110/66, respiratory rate 18, pulse oximetry revealed an oxygen saturation of 100% on room air. Weight was 56.7kg, height 65 inches. HEENT was normal. Lungs were clear to auscultation. Heart had a normal S1 and S2 without any appreciable murmurs, rubs or gallops. Abdomen was soft, non-tender, non-distended with active bowel sounds and no hepatosplenomegaly. Her extremities were all normal, except for her right upper arm and shoulder which was diffusely swollen with distended veins noted over the upper arm and shoulder. No erythema was noted. She had decreased sensation to gross touch over her entire right arm compared to the left. Radial pulses were strong and equal bilaterally. She was diffusely tender to palpation over her right upper arm and

shoulder. She had full range of motion of her right arm, but stated there was pain as she abducted her arm beyond ninety degrees. Strength and DTRs were normal. No supraclavicular, cervical or axillary lymph nodes were palpable. Measurements of her right and left arms are as follows: mid forearm R 19cm, L 18cm; distal upper arm R 24cm, L 23.5cm; mid upper arm R 27.5cm, L 23.5cm; proximal upper arm R 25.5cm, L 23cm.

PA and Lateral chest radiographs were obtained to evaluate the presence of a lung mass, anatomic abnormalities or any other cause of lymphatic obstruction and was normal. CBC was normal except for mildly elevated platelets of 409. LDH, CK, ESR and CRP were all normal. Urinalysis was normal.

After a thorough discussion with the radiologist on call, it was determined that the best course of action to rule out a clot or lymphatic obstruction was to consult vascular surgery. The patient was then scheduled for a Doppler ultrasound of her right arm the next day with follow up with vascular surgery.

The Doppler ultrasound revealed thrombosis within the right axillary and brachial veins which was suggestive of thoracic outlet syndrome. After evaluation by vascular surgery, subclavian vein effort thrombosis, also known as Paget-Schroetter Syndrome, was suspected. She underwent right subclavian venogram under conscious sedation which revealed a near occlusion of the subclavian vein where it passed under the right clavicle. Numerous collaterals were noted to have developed, reconstituting the subclavian just before it joined the right internal jugular vein. She was then admitted to the Intensive Care Unit for t-PA infusion at 1mg/hr. Five hours later, a venogram was repeated which revealed some improvement in the appearance of the clot/web. The loose clot was cleaned out with

angio-jet and the following venogram showed a small channel through the clot. The patient was then placed in an Addison's maneuver and the venogram was repeated. This showed no flow through the affected area of the subclavian vein. This confirmed their diagnosis of effort thrombosis of the right subclavian vein. Angiography revealed a web stenosis at the junction of the first rib and clavicle. She will be scheduled for a first rib resection at a later date.

Discussion

Paget-Schroetter syndrome, also known as spontaneous axillosubclavian vein thrombosis or effort vein thrombosis, is a relatively rare occurrence. Estimated incidence is 2 per 100,000 persons per year. Primary thrombosis of the axillary subclavian vein was first described in 1875 by Sir James Paget and again in 1884 by Leopold Von Schroetter. In the 1960s it was coined "effort vein thrombosis" in order to acknowledge the role of unusual exertion of the arms causing this phenomenon.

There are a number of causes that have been theorized, but the paucity of cases has made it difficult to confirm. Cases in the literature have pointed to a temporal and causal relationship of repetitive shoulder-arm motion, particularly retroversion or hyperabduction of the arm preceding the development of symptoms. Young healthy persons such as golfers, weightlifters, baseball players, tennis players, cheerleaders, painters and beauticians seem to be more prone to developing thrombosis of their deep vein in their dominant arm. The theory is that an anatomic anomaly of the clavicle or first rib causes a compression of the subclavian vein when the arm is in lateral abduction resulting in microtrauma to the vessel intima which then leads to the activation of the coagulation cascade.

Anatomic strictures of the thoracic outlet caused by hypertrophy of the tendon of the subclavian muscle or anterior scalene muscle, fibromuscular bands or a callus from an old clavicle fracture can also compress the vein against the first rib. Hypercoagulable states to include exogenous estrogens in females can increase the risk of developing this condition. In the case that was presented, this healthy, young, adolescent female revealed that she was a relay track runner who frequently retroverted and laterally abducted her right arm to receive the baton from her teammate.

When a young healthy individual presents with signs and symptoms of venous obstruction, it is prudent to perform ancillary tests to rule out Paget-Schroetter syndrome. Symptoms often consist of a dull, aching pain in the shoulder and/or axilla that worsens with exercise with physical signs of swelling, bluish discoloration and venous collaterals. Ultrasonography with color doppler is a non-invasive test with good sensitivity and specificity to identify thrombosis of the axillary subclavian vein. Ultrasound, however, provides little data if surgical intervention is required. Contrast venograms are required to evaluate the extent of the thrombus. A plain film of the chest and cervical spine can identify anomalies if the clavicle, first rib, cervical ribs, long transverse processes of the cervical spine or musculofascial bands that could be compressing the vein. There is little data regarding the use of CT or MRI/MRA for the diagnosis of upper extremity vein thrombosis. Although specificity of these tests is good, the sensitivity has not proven to be adequate as a screening tool. This patient was screened with a Doppler ultrasound and underwent venography to define the extent of the lesion prior to undergoing therapy.

Once the diagnosis of Paget-Schroetter is made, treatment to

minimize long term sequelae must be instituted. Case reports in the literature identify anticoagulation, thrombolytic therapy, and surgical treatment or a combination of these three as acceptable options. The more commonly accepted therapy that minimized long term morbidity is catheter-directed thrombolytic therapy with tissue plasminogen activator (t-PA) followed by outpatient anticoagulation with low molecular weight heparin or warfarin for three to six months. The patient should undergo follow up ultrasound and venography to document patency of the vessel. These studies should be performed both in the neutral position as well as with the affected shoulder abducted and retroverted to identify possible causes of venous obstruction. Surgical options include balloon dilation of the venous stenosis, however, the compression is often from an external source therefore is not useful. Surgical decompression of the thoracic outlet by removal of the first rib is helpful, using stents as an alternative treatment. The patient in this case underwent thrombolytic therapy with t-PA, anticoagulation with heparin as an inpatient then scheduled for a first rib resection at a later date after a web stenosis between the clavicle and the first rib that was identified on venogram.

Complication rates vary among the literature. The most common is embolic complications such as pulmonary embolism whose incidence ranges from 10-30 percent with the majority of these remaining asymptomatic. Another common cause of morbidity is post-thrombotic syndrome which consists of venous hypertension secondary to outflow obstruction and valvular injury. Symptoms can be as mild as mild edema to incapacitating limb swelling with pain and ulceration.

Patients who are identified as having Paget-Schroetter syndrome should undergo a further workup to

identify potential causes of a hypercoagulable state. Blood should be drawn to evaluate for Factor V Leiden, Protein C and S, antithrombin III, plasma homocysteine, antiphospholipid antibodies, prothrombin gene mutation and anticardiolipin antibody. A good history should be taken to identify exogenous use of estrogen, unexplained recurrent pregnancy loss or a personal history of previous DVTs. Treatment should also be directed toward these causes of hypercoagulable states to prevent future occurrences.

In summary, physicians who see healthy, active children and adolescents should be aware of Paget-Schroetter Syndrome despite its rare occurrence. If treated promptly, long term morbidity and mortality from embolic events and permanent structural vessel damage can be prevented or minimized.

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specific changes for the adolescent.

When addressing STI's with teens, it is necessary to recognize that infection with the Human Papilloma Virus (HPV) is one of the most common STI's with a reported prevalence ranging from 13-50% in sexually active young females.^{1,4} Several biologic and behavioral risk factors may make young females more vulnerable to acquiring an HPV infection. Additionally, evidence supports the strong causal relationship between HPV and abnormal changes on Pap smear cytology.¹

To date more than 100 different types of HPV have been identified, of which 30 have a predilection to invade the genitourinary tract. HPV is divided in to low risk and high risk types. The high risk HPV types

cause the greatest concern as they are strongly associated with the development of cervical cancer.¹ The system of grading pap smears has undergone many changes over the years. The current system is the Bethesda System (updated 2001) that grades pap smears on the degree of severity of abnormal changes on the cervix. The listed categories include: within normal limits, Low-grade Squamous Intraepithelial Lesion (SIL), Atypical Squamous Cells of undetermined significance (ASC-US), Atypical Squamous Cells – cannot exclude High-grade (ASC-H), and High-grade SIL.^{1,2} Generally Low-grade SIL and ASC-US are felt to be more benign lesions.¹

The new liquid preparation for cervical cell sampling has several advantages versus the traditional method of fixing the sample on the slide. The Liquid based sampling allows better fixation and therefore better analysis of the cells. In addition, HPV DNA typing can be performed. This specific information on high versus low risk HPV type will help guide the decision making for management of abnormal paps.^{2,3}

As the prevalence of HPV has risen in adolescent females, so has the prevalence of abnormal pap smears. Studies of adolescent females describe rates varying between 3-17%.² Fortunately, studies have shown the majority of HPV infections resulting in abnormal paps are predominantly in the low risk categories of Low-grade SIL and ASC-US (8.8 and 12.2% respectively) compared to High-grade SIL (0.7%).¹

Previous guidelines required colposcopy and potential biopsy and/or treatment for all types of pap abnormalities with the exception of ASC-US.² Colposcopy can be an uncomfortable and even painful experience particularly for a teenage female. From new understanding of

the natural course of an HPV infection, it is understood we are over-diagnosing and over-treating many patients. Moscicki et al in their studies of HPV positive adolescent females has been able to map out the natural course of infection. She has shown that 75% of those with HPV infection will clear by 30 months without intervention. More importantly, 90% of those with low risk HPV type infections will regress. Even those with high risk type infections, 70% will regress, and only a small percentage progressed to High-grade SIL.³ These findings are guiding the new direction of recommendations to decrease the amount of invasive management for abnormal pap smears.

What are the new recommendations? First, screening had been initiated after the first sexual intercourse experience or by age 18 years, whichever came first. Because of the strong causal relationship of HPV with abnormal changes on the cervix, there is little need to initiate screening for a female who remains abstinent until a later age. The incidence of invasive cervical cancer is negligible until a female reaches her early twenties. Also influencing the new recommendations is the understanding that the majority of the pap abnormalities will regress within 30 months with a low likelihood of progression. Therefore the new guidance on initiating screening is to begin within 3 years of sexual debut or by age 21 years, whichever comes first.^{5,6}

Consistent with the old standards, any High-grade SIL requires referral to Gynecology for colposcopy to establish the degree of abnormality and treatment of the identified lesion. Pap results indicating ASC-H also require referral. 15% of those labeled ASC-H will actually be a high-grade lesion. In this case, it is best to have

the colposcopy exam to aide the diagnosis and treatment of this lesion.^{2,5,6}

Many of the new changes are reflected in the management of Low grade SIL and ASC-US. An acceptable alternative for managing both of these diagnoses is to repeat screening at 6 and/or 12 months. If the abnormality persists at the 12 month follow up, perform HPV DNA type testing. If the HPV DNA testing is positive for high risk types, then refer the patient to Gynecology for colposcopy. Persistent Low-grade SIL and ASCUS with low risk type HPV do require referral for colposcopy exam. Persistent is generally defined as two abnormal pap results. Specific guidance on pap smear screening in adolescents is still an ongoing process. New data supports that adolescent females are at high risk of acquiring HPV infections. Fortunately, it is clear that the majority of females have low risk type infections, few progress to High grade SIL, and cervical carcinoma is very uncommon. Although screening has made a tremendous difference in cervical cancer rates and mortality, it appears that for adolescent females less invasive intervention is warranted.

This does bring to light an additional factor: how good are adolescents patients at following up? Several studies have shown that despite good intention to return, compliance was low. Multiple variables are associated with this finding, but pediatricians have the opportunity to positively impact on this behavior. Utilizing clinic visits to address gynecologic health, and ensuring the young female patient has a positive understanding regarding the importance of cervical cancer screening and following up results.⁷ Part of preventive health care for the young female is addressing sexual activity to promote risk behavior reduction, but also to

educate on the importance of gynecologic health.

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