Welcome to the Philippine Heart Center, Division of Pulmonary and Critical Care Medicine’s interhospital case presentation. To start our program, we’ll have the invocation which will be led by Dr. Angelene Taleon and to be followed by the Philippine national anthem, PCCP hymn and the PHC hymn to be led by Dr. William Del Poso.

You may now be seated. To give his opening remarks may we call on the Head of the Division of Pulmonary and Critical Care Medicine of the Philippine Heart Center, Dr. Fernando G. Ayuyao

In behalf of the Division of Pulmonary and Critical Care Medicine of the Philippine Heart Center and the Philippine College of Chest Physician, we are indeed very happy to welcome all of you to this interhospital symposium. I would like to recognize some of the dignitaries that we have, of course tonight we have our reactors, first Dr. Ferdinand Bernal, Dr. Myla Verastigue–Custodio, of course our past President of the PCCP, Dr. Maricar Blanco–Limpin. We have of course our past President and Chairman Emeritus, Dr. Teresita S. De Guia and other colleagues in the Philippine College of Physicians, co-fellows and co-students who want to learn more in medicine, friends, ladies and gentlemen.

Such a night like this, it always give me a nostalgic feeling probably because this started in 1983 when a young gentleman from this institution started what was called a case presentation among hospitals and he presented a case of recurrent of thromboembolism – and this is the guy who had the first vena caval filter that was inserted to this patient. That was a reported case during that time and indeed that guy is the one talking in front of you and through the years we have evolved. The last interhospital in 1999, I was the president of the Philippine College of Chest Physicians and the first interhospital in the year 2000, I was still the president of the Philippine College of Chest Physicians, so really, it does give me a lot of feeling of being within this kind of interhospital symposium while Dr. Obillo-Mison, I forgot the past president, my Madre Choli Obillo–Mison was my colleague when we presented during that time. While during that time we were also still using this kind of boards, I was using a projector and sometimes, the slides are very thick and the projector did not like to move, at times we will be using acetate in cases when we need some more data to be presented but now with the click of your fingers you can have all your data available and you can easily change what is in the text of your
discussion. But while it is true that everything seems to be easy in your fingers now it is much more difficult to present in this august body.

Today we are going to present a very interesting but a very complicated syndrome. The aim of the division is to perfuse your brain so that you will not fall asleep. We will put iron bars on your back so that you will not strain, you will not slouch, more so you will not increase the curvature of your spine. We will give you huge food for your brain so that it will swell and become obese. And we hope in the end of this symposium you have learned a lot from us. On behalf again of the division of the Philippine Heart Center and the Philippine College of Chest Physician, we welcome you all and thank you.

DR. ABARA: Thank you Dr. Ayuyao. Now moving on to the main event of this evening’s symposium, the presentation of this very interesting case. This will be presented by our senior fellows: Dr. James Albert Flores will discuss on the history, physical examination and the differential diagnosis, to be followed by Dr. Hygeia Mampao who will elucidate on the diagnostics then by Dr. Dehuel Cuyacot who will discuss on the pathophysiology and lastly by Dr. Ethel Cabrera who will discuss on the management. Friends and colleagues let us welcome our presentors.

The Presentors: Dr. Dehuel Cuyacot, Dr. Hygeia Mampao, Dr. Ethel Cabrera, Dr. James Albert Flores (from left to right)

VIDEO PRESENTATION: Dr. William Del Poso (voice over)

The association between obesity and hypersomnolence has long been recognized. We know of Joe, the fat boy, from the work of Charles Dickens, The Pickwick Papers, who was markedly obese and tended to fall asleep uncontrollably during the day. The understanding on obesity hypoventilation syndrome has increased since Auchincloss first described the syndrome in 1955 eventually being known as Pickwickian syndrome.
Did Quasimodo, the legendary hunchback of Notre Dame, have serious sleep disturbances that could explain certain aspects of his personality? Victor Hugo never resolved the question. It was not until 1981 that this was answered - when Guilleminault described how severe kyphoscoliosis resulted in alterations in sleep states, coining the term Quasimodo syndrome.

Fast forward to present, these syndromes collide, we have a new face nobody has seen before, obesity, kyphoscoliosis and obstructive sleep apnea all occurring in a single patient. We have a ménage a trois. A threesome so to speak of three distinct disease entities overlapping to cause profound pathologic alterations.

Tonight, we will have the opportunity to study a patient with a preternatural mix of severe obesity, severe kyphoscoliosis and severe obstructive sleep apnea. Welcome and good evening.

**Dr. James Albert Flores:** The following are the objectives of this presentation: to present a case of a 29 year-old male with kyphoscoliosis, obesity, and obstructive sleep apnea; to discuss the approach in the diagnosis; to describe their anatomic and physiologic consequence; to present the treatment options; and to elucidate a new syndrome where kyphoscoliosis, obesity, and obstructive sleep apnea all present in a single patient.

PD is a 29 year-old, male, known hypertensive, known diabetic, morbidly obese, with kyphoscoliosis. He was referred to our institution for polysomnography.

PD had an eight year history of... gradual weight gain, with an increased appetite and decreased physical activity, increased sleeping time, lasting more than 10 to 12 hours at night, loud snoring, witnessed apneic episodes, lasting 10 to 20 seconds, increased daytime sleepiness followed despite extended hours of sleep. Other associated symptoms were... morning headaches, constant irritability, mood swings and memory lapses. He exhibited poor academic performance, constantly failed in class and eventually dropped out.

Four years prior to admission, there was continued weight gain, with persistent fragmented and nonrestorative sleep. Excessive daytime sleepiness manifested as having more frequent naps, had several occasions of sleeping in the bus and missing his stop; and had episodes of falling asleep even while standing. The memory lapse manifested as being unable to sustain long conversations and was very forgetful.

Two years prior to admission, he was apparently treated as a case of community-acquired pneumonia high risk. During this admission, he had hypoxemia and hypercapnia, and was intubated and on mechanical ventilatory support. He was discharged improved. A consideration of Obstructive Sleep Apnea was made and was advised work up did not comply. The same scenario occurred six months prior to admission.
One month prior to admission, he was again readmitted due to pneumonia, presenting as productive cough and difficulty of breathing. Laboratory exams revealed increased hemoglobin and hematocrit; normal thyroid function tests. Echocardiography revealed normal pulmonary artery pressure, and normal RA, RV and main pulmonary artery dimensions, ejection fraction of 65%, with eccentric LV hypertrophy. He was discharged and was referred to our institution for polysomnography.

Significant past medical history revealed, hypertension and diabetes, childhood medical history revealed, meningocele at birth; had meningocele repair surgery at 4 months; Diagnosed with kyphoscoliosis at 8 years, workup done; advised surgery but did not comply; No history of accidents, serious physical injuries or trauma

The patient's father is hypertensive, and both his parents and siblings are obese. He is a previous six pack year smoker, quit 4 years ago and an occasional alcoholic beverage drinker.

The review of systems revealed easy fatigability, dyspnea on exertion, chest pain

The patient is morbidly obese with a BMI of 40.8 kg/m$^2$, with the following vital signs. He has a short neck, neck circumference of 48 centimeters or 18.9 inches (Figure 1); normal sized tongue with no retrognathia, Mallampati 4 with only the hard palate is visible (Figure 2).

Angulated shoulders with hip tilt (Figure 3), asymmetrical chest expansion with lagging on the left hemithorax, prominent rib hump at right posterior chest, exaggerated by forward bending (Figure 4), percussion was technically difficult, vesicular breath sounds on both lung fields.

Heart examination revealed an accentuated P2. Abdomen is globularly enlarged, waist circumference of 140 cm or 55.1 inches (Figure 5).

No edema, no cyanosis, no clubbing, hyperpigmentation of the distal aspect of both legs, Arm span 168 cms (66.1 inches) which is a surrogate of his true height with an arm span to height ratio: 1.1.

Neurologic examination was unremarkable.

With that presented, we had an admitting impression of Obstructive Sleep Apnea, Kyphoscoliosis, and Morbid Obesity. Given the history and physical examination, we considered a sleep related breathing disorder.

Sleep related breathing disorders are characterized by abnormal respiration during sleep. The three major sleep related breathing disorders are Central sleep apnea syndromes, Obstructive sleep apnea syndromes, and sleep related hypoventilation/hypoxemia syndromes.

Central sleep apnea syndrome is characterized by a lack of drive to breathe during sleep, resulting in insufficient or absent ventilation and compromised gas exchange. The following are the types of central sleep apnea... Obstructive Sleep Apnea Syndrome is a disorder that is characterized by obstructive apneas and hypopneas caused by repetitive collapse of the upper airway during sleep. Types include the following... The third category is sleep related hypoventilation-hypoxemia syndromes. Variants include the following...
We considered obstructive sleep apnea, obesity-hypoventilation syndrome, central sleep apnea, and sleep disordered breathing due to kyphoscoliosis.

Next we will compare and contrast the three main considerations... OSA, OHS, and CSA. Generally speaking, the three present similarly. With their symptoms mainly related to disrupted sleep. Nocturnal symptoms are the following (Figure 6). Checked are those seen in our patient. Daytime symptoms are enumerated (Figure 7). Checked are those seen in our patient.

With most symptoms similar to OSA, OHS patients commonly present with symptoms of pulmonary hypertension and right sided heart failure. Dyspnea on exertion is a clue that OHS is present because patients with OSA - alone generally do not develop dyspnea on exertion. Next is central sleep apnea, the most common reported symptoms are insomnia and excessive daytime sleepiness. In general, the degree of daytime hypersomnolence is less than that observed with obstructive sleep apnea, and insomnia is more prominent. Patients with secondary CSA also have features of the underlying disease.

We move on to physical examination, OSA patients commonly show the following. PD was obese, a neck circumference of 18.9 inches, mallampati 4 and hypertensive. In OHS, physical examination shows plethoric obesity, enlarged neck circumference, crowded oropharynx, and prominent pulmonic component of the S2. All of which are seen in our patient. For central sleep apnea, no physical findings are predictive and patients usually have a normal body habitus

For the risk factors, OSA has definite and potential risk factors. Obesity is the best documented risk factor. Craniofacial and upper airway soft tissue abnormalities, current smokers, but not past smokers, and diabetes. Our patient was obese, Mallampati 4 and is diabetic. OHS, shares most of the risk factors of OSA, with obesity being the main risk factor. For central sleep apnea, risk factors are increased age, male gender, heart failure and stroke.

Another consideration is sleep disordered breathing secondary to a chest wall disorder. Our patient has kyphoscoliosis. Sleep related breathing disorder among them commonly present as obstructive sleep apnea or nocturnal hypoventilation. Daytime respiratory symptoms are more common when the angle of the spinal deformity approaches 100 degrees.

We move on to the diagnostic criteria. The diagnosis of OSA is confirmed if the number of obstructive events, apneas, hypopneas or respiratory event related arousals on PSG is greater than 15 events per hour or greater than 5 per hour in a patient that reports any of the following symptoms (Figure 8).

OHS is diagnosed when the following criteria are confirmed: Obesity or BMI of more than 30, awake alveolar hypoventilation that cannot be attributed to other conditions such as pulmonary disease, skeletal restriction, neuromuscular weakness, hypothyroidism, or pleural pathology (Figure 9). The absence of an alternative cause of hypoventilation is an important requirement for the diagnosis of OHS. For central sleep apnea, the individual must fulfill A, B, and C for a diagnosis (Figure 10).
With that presented, the differential diagnoses cannot be ruled out by the patient’s history and physical examination. For findings invariably overlap among the considered forms of sleep related breathing disorders.

Polysomnography is essential. However, other workup is necessary, Arterial blood gas testing to document hypercapnia and or hypoxemia, a high serum bicarbonate level is a clue of chronically hypercapnia. Pulmonary function tests for evidence of obstructive or restrictive disease. Radiographs to look for parenchymal lung disease and chest wall disease. Echocardiography to document right sided heart failure and/or pulmonary hypertension.

The diagnostic examinations, their results and implications will be discussed by Dr. Hygeia Mampao.

Dr. Hygeia Mampao: Thank you very much Dr. Flores. We are presented with a 29 year old male with daytime somnolence, known hypertensive and diabetic. Examination revealed an increase in neck circumference, Obese 2, and Mallampati 4. All of these are features suggestive of Obstructive Sleep Apnea. Obesity, hypertension, and Type 2 diabetes puts at him as High Risk for OSA as stated in the latest AASM Guidelines. From the same article, patients deemed high risk should have the diagnosis confirmed and severity determined with objective testing.

The patient was evaluated for his daytime sleepiness through Epworth Sleepiness Scale. He had a score of 19 out of a maximum 24 which means that the patient has excessive daytime sleepiness and opted him to undergo polysomnography (Figure 11).

Polysomnography is a diagnostic test used in the evaluation of sleep disorders. Various physiological sensors are connected to the patient. Sensors on the face and scalp measure eye movement and brain activity. Sensor at the nose to measure airflow. Sensor on the finger measures the amount of oxygen saturation. Elastic belt sensors around chest and abdomen measures amount of effort to breathe. Wires transmit data to the computer generating an Epoch.

Showing you a 120 second epoch. Zooming into a 30 second epoch in REM stage of sleep characterized by rapid eye movements in electro-oculogram. In this epoch an obstructive apnea was noted. Defined as a drop in peak signal excursion by >90% of pre event baseline, in duration of more than or equal to 10 seconds. Hypopneas were also noted which is defined as Peak signal excursions drop by more than or equal to 30% of pre-event baseline, with duration of more than or equal to 10 seconds, and with more than or equal to 3% oxygen desaturation from pre event baseline. During this period, desaturation was also noted. Our patient was noted to have hypopnea, apnea, and desaturation (Figure 12) with an Apnea-Hypopnea Index of 59.8 per hour of sleep which according to AASM classification denoted a severe state. Hence, he was signed out as a case of Severe Obstructive Sleep Apnea-Hypopnea Syndrome.

Therapeutic Study was done. CPAP at pressure of 17 cmH$_2$O abolished apnea and hypopnea. Therefore, nightly use of CPAP at 17 cm water was recommended.
Another remarkable physical finding was the presence of prominent rib hump on the right posterior chest (Figure 4). An x-ray of the thoracolumbar spine showed a rightward deviation with T4 as the upper end vertebra and L3 as the lower end vertebra yielding a Cobb angle of 100 degrees (Figure 13). He was diagnosed with severe kyphoscoliosis.

Kyphoscoliosis is a disease of the spine and its articulations. The deformation of the spine consists of kyphosis which is an anteroposterior angulation of the spine and scoliosis which is lateral displacement of the curvature. It occurs 1 in 1000 for mild deformity to 1 in 10,000 for severe spinal deformity. This is a representation of how Cobb angle is measured. It is used to determine the prognosis, predicts the effects of disease to lung function, and the risk of developing respiratory failure.

Arterial Blood Gas showed, Hypoxemia with $pO_2$ of 56, oxygen saturation of 85.6%, hypercapnia with $pCO_2$ of 66.5, and bicarbonate of 36, interpreted as chronic Respiratory Acidosis with Moderate Hypoxemia.

Rom and associates presented patients with severe kyphoscoliosis that showed hypercapnia with mild to moderate hypoxemia. Comparing his subjects with our patient, our patient is much younger at age 29 with a more deranged hypercapnea and hypoxemia. This led us to think whether the kyphoscoliosis is aggravated by sleep disorder of this patient.

A study by Mezon and associates, presented 5 patients with kyphoscoliosis and sleep disorder. However, his subjects presented with Central Sleep Apnea in contrast to our patient who presented with Obstructive Sleep Apnea. Mezon’s subjects showed that during REM sleep, physiologic disturbance was greatest, being the time of greatest desaturations. This proves to be the same with our patient.

Another study made by Guilleminault presented 5 patients with kyphoscoliosis and Obstructive Sleep Apnea. He coined the term Quasimodo Syndrome. His subjects were older with weight ranging 57-71 kgs compared to our patient who is much younger at age 29 and heavier with weight of 92kg. He found his subjects to have a predominantly obstructive type of apnea which is consistent with our patient. Similar to Guilleminault’s finding, our patient has lowest oxygen saturation during REM sleep. However, comparing the degree of hypoxemia and hypercapnea, our patient has more deranged values than those found in Guilleminault’s subjects. Is this because our patient is also obese? How will obesity affect the respiratory function? With the presence of obesity, should we consider him still as a case of Quasimodo Syndrome? On further work-up.

CBC showed polycythemia which could be secondary to previously mentioned hypoxemia. Chest x-ray showed a right lung base subsegmental atelectasis and marked thoracolumbar dextroscoliosis (Figure 14) as previously discussed.

Pulmonary Function test was done with a flow-volume loop narrowed due to decrease lung volume. The FEV1/FVC ratio was normal, FVC and FEV1 (Figure 15) were decreased suggestive of a probable Moderately severe restrictive ventilatory defect. Lung volume study done showed decreased total lung capacity, vital capacity and FRC (Figure 16), confirming the finding of Moderately severe restrictive ventilatory defect. Diffusion study is moderately reduced. These findings are consistent with those found in patients with kyphoscoliosis.
A study by Caro and associates showed 38 kyphoscoliotic patients with restrictive ventilatory defect. All have decreased total lung capacity, vital capacity, and functional residual capacity. Hoffstein, on the other hand found no direct relationship between pulmonary function studies and Obstructive sleep apnea. Hence, the findings of severe restrictive ventilatory defect in our patient was primarily due to kyphoscoliosis.

Echocardiography showed an EF of 65%, normal LV dimension with good wall motion and contractility, High normal RV dimension, normal Main Pulmonary Artery, and Pulmonary Arterial Pressure. Is this an early finding of increased RV workload that would eventually result to Cor Pulmonale?

Our patient is hypertensive, diabetic, with central obesity and hypertriglyceridemia, satisfying the diagnosis of Metabolic syndrome.

Despite substantial evidence from several studies to suggest an independent link between Obstructive Sleep Apnea and Metabolic Syndrome, the issue still remains controversial. A study made by Lam and associates among Chinese volunteers showed an association with OSA and Metabolic Syndrome. He found that OSA has five-fold risk of having metabolic syndrome. So, is there really a connection between Obstructive Sleep Apnea and Metabolic Syndrome?

A study made by Palaniappan showed that Obesity is main precursor of the Metabolic Syndrome. From the study of Young, obesity is also a significant factor in the development of Obstructive Sleep Apnea. These studies prompted Parish and associates to hypothesize Obesity as the association between OSA and Metabolic Syndrome.

The BMI cut points from the 1998 WHO Consultation on Obesity are generally accepted, however the International Obesity Taskforce developed cut-off points appropriate for Asians. Thus, based on that classification, our patient is categorized as Obese II.

With the data at hand, we think: Is this a case of Obesity and obstructive sleep apnea giving us Obesity hypoventilation syndrome? However with Kyphoscoliosis coming into the picture, does it rule out obesity hypoventilation syndrome? Or does it make this case a Quasimodo Syndrome? If you recall, they do not present with obesity making our case even more unique (Figure 17). To give us the dynamics and bridge the gaps in this ménage à trois of Obstructive Sleep Apnea, Kyphoscoliosis and Obesity here is Dr. Cuyacot.

**Dr. Dehuel Cuyacot:** Thank you Dr. Mampao. The ménage à trois of the three diseases in a single individual will come to a common end point. To bridge the gaps, we must first discuss each entity by itself. It all started with the patient having severe kyphoscoliosis.

The rotation of the spine disrupts the respiratory mechanics by decreasing the chest wall compliance and increasing elastic recoil of the chest wall, thereby decreasing the lung compliance. This results to a restrictive ventilator pattern as shown by the decreased TLC, VC, FEV1, FVC with a normal FEV1/FVC ratio, also presenting as micro-atelectasis, as seen in our patient (Figure 18).
In adults, the severity of the restrictive process is directly proportional to the severity of the spinal deformity. The relationship can be computed from the following equation, yielding a predicted vital capacity (Figure 19).

Thus curves of 10 to 25 degrees are considered mild, with a decline in predicted vital capacity of 84 to 79%. Curves of 25 to 45 degrees are considered moderate with a decline in predicted vital capacity of 79 to 72%. Curves of more than 45 degrees are considered severe with a decline in predicted vital capacity of less than 72%. However, starting at 65 degrees, V/Q mismatch occurs, leading to hypoxemia. At angles more than 100 degrees, respiratory failure occurs. Our patient has a Cobb angle of 100 degrees with a predicted vital capacity of 46, which may account for the recurrent hospital admissions due to respiratory failure.

Our patient was later diagnosed with obstructive sleep apnea. Can kyphoscoliosis affect sleep? Is there a connection?

In kyphoscoliosis, the stiffened chest wall leads to increased elastic load leading to a heightened respiratory drive, so that diaphragm activation increases, also there is greater recruitment of the inspiratory muscles of the rib cage. When the patient goes in to non-REM sleep, the neural drive to the intercostal muscles is diminished and may be absent in REM sleep. Hence the burden of expanding the non-distensible chest wall falls more on the diaphragm, thereby causing hypotonia leading to hypoventilation (Figure 20).

Inspiratory activation of upper airway muscles occurs earlier than activation of the diaphragm, which stabilizes the upper airway and counterbalances the collapsing force exerted on the upper airway by diaphragm. Any reduction or delay in upper airway inspiratory muscle contraction, relative to diaphragm activity, predisposes to upper airway narrowing or collapse during sleep.

The combination of kyphoscoliosis and obstructive sleep apnea is Quasimodo syndrome as coined in the study of Guilleminault as presented earlier. He attributed the OSA to the collapse of the upper airways to the mechanism previously discussed.

On the other hand, a study done by Al-Kattan and associates using bronchoscopy in kyphoscoliotic patients showed that OSA and nocturnal hypoxemia could be due to the secondary twisting of the extra-thoracic trachea (Figure 21). Unfortunately, bronchoscopy was not done in our patient.

So to answer the first question of Dr. Mampao, does our patient have the complex of kyphoscoliosis and OSA called the Quasimodo syndrome? The answer is yes. However, as mentioned earlier, our patient presented much earlier than expected. Furthermore, a more deranged hypercapnea and hypoxemia were noted compared to the subjects in the study. Could there be another factor that is causing the earlier symptoms, resulting to a more deranged clinical picture?

We are dealing with a patient who has morbid obesity and OSA. These two can work in concert to produce a third disease entity, Obesity Hypoventilation Syndrome.
A study by Mohkhlesi showed a close relationship between BMI and OHS. As the BMI increase to 40 and above, the likelihood of OHS increases in parallel (Figure 22). Likewise, 90% of OHS patients also have OSA.

In OSA, post-apneic hyperventilation after arousal in an important factor for decreasing PCO$_2$ and increasing oxygenation. However, patients with OHS are unable to normalize their PCO$_2$ between such respiratory events.

With the failing pH, the kidney buffers by decreasing bicarbonate excretion, and serum bicarbonate accumulates. Eventually the serum bicarbonate level becomes high enough to depress ventilation, causing chronic hypoventilation and hypercapnea (Figure 23) both during sleeping and waking hours. This explains the blood gas of our patient.

So does our patient have obesity hypoventilation syndrome? The answer to the question would have been straightforward. We cannot fulfill all the criteria for OHS due to the presence of kyphoscoliosis.

The absence of an alternative cause of hypoventilation is an important criterion for the diagnosis of OHS. Patients may have other cause of hypoventilation such as obstructive airways disease, interstitial lung disease, chest wall disorders such as kyphoscoliosis, hypothyroidism, and neuromuscular diseases. If the other disease is mild and unlikely to cause hypercapnea, then it is reasonable to give the patient a diagnosis of OHS. But if the other disease is more severe and probably contributing to hypercapnea, the situation becomes more complicated. Thus, in this setting, pure OHS cannot be diagnosed with certainty. However, in a patient with severe obesity, severe sleep apnea and oxyhemoglobin desaturation during sleep, we generally presume that OHS may be a component of the disease complex of our patient.

By the same token, there is a well-established relationship between OSA and obesity. Obesity results in anatomic and functional changes like decreasing pharyngeal airway size, and increased leptin resistance that can result to the development of OSA in obese individuals. Likewise, OSA may predispose individuals to worsening obesity because of sleep deprivation, daytime somnolence and disrupted metabolism (Figure 24).

In OSA, the intermittent hypoxia and reoxygenation also induces the production of reactive oxygen species resulting to endothelial dysfunction thereby promoting systemic inflammation, causing cardiovascular events, impaired fasting glucose, and hypertriglyceridemia leading to Metabolic syndrome - which manifested in our patient. This vicious cycle explains the role of obesity in OSA and metabolic syndrome as mentioned by Dr. Mampao.

As to the connection between obesity and kyphoscoliosis, a thorough search of the literature yielded no published data.

Looking now at the bigger picture. our patient has an ominous combination of obesity, obstructive sleep apnea and kyphoscoliosis. What would we name this syndrome? This merger of three diseases is indeed complex and unique and would require a multi-disciplinary management, which will be presented by Dr. Cabrera.
Dr. Ethel Cabrera: Thank you Dr. Cuyacot. The ménage a trois of obesity, kyphoscoliosis and obstructive sleep apnea requires a long-term, multidisciplinary approach to management. Let us first discuss the management options of kyphoscoliosis. PD was diagnosed at 8 years old and was previously advised surgery but did not comply. The present state of his kyphoscoliosis is severe, with the angles of scoliosis and kyphosis both at 100 degrees.

Operative treatment traditionally consists of spinal fusion and or insertion of rods with goals of prevention of curve progression and partial curve correction. Spinal surgery should be performed at an early age, ideally before the age of skeletal maturity, and it is rarely effective in adults. In a study by Tzelepis, spinal surgery in patients more than 20 years old did not result in significant improvement of vital capacity or gas exchange and posed a high, 20 percent complication rate.

With our patient presenting with severe kyphoscoliosis at 100 degrees, and the presence of his comorbidities, he is not a good candidate for spinal surgery. Hence, supportive therapy should be maximized. Through immunizations – due to their predisposition to recurrent infections, supplemental oxygen for hypoxemia and for correction of polycythemia, adherence to smoking cessation, and weight reduction. Specific nonsurgical approach include, noninvasive ventilation and pulmonary rehabilitation.

Indications for initiating NIV include, hypoventilation, hypercapnia, and oxyhemoglobin desaturation. All of which were present in our patient.

Long term oxygen therapy combined with positive pressure ventilation in kyphoscoliotic patients results in significant increases in vital capacity, maximal inspiratory pressure, arterial oxygen and a decrease in carbon dioxide. Shown are the survival curves of kyphoscoliotic patients on long term oxygen alone versus long term oxygen plus positive pressure ventilation. After 1 year of treatment, survival was only 66% in the LTO group versus 100% in the NIPPV group (Figure 25).

Pulmonary rehabilitation can bring about improvements in patients with restrictive lung disease. A study by Bihiyga of 31 patients, 14 of which had kyphoscoliosis, a 24 week program showed significant improvements in exercise capacity, muscle force and scores on the chronic respiratory disease questionnaire.

In another study by Ong-Cabrera and associates in our institution among non-COPD patients, an 8 week program showed improvements in exercise capacity. With the following improvements on 6 minute walk test distance and perceived breathlessness and muscle fatigue.

On the other hand, obesity and OSA are best approached in tandem.

The consensus initially focuses on patient education and support on the following points. It is in the process of education that interventional therapy can be introduced, such as positive pressure airway therapy or PAP, the primary treatment for OSA.
Positive airway pressure therapy is the most effective treatment for OSA. It can be given in the form of continuous positive airway pressure or CPAP, the gold standard. It is indicated for moderate to severe OSA and for those with comorbidities. CPAP was initiated in our patient. Another form of PAP is bilevel positive airway pressure or BiPAP, used in patients requiring higher pressures and for those intolerant to CPAP. Lastly, the auto-titrating positive airway pressure or APAP, used in moderate to severe OSA with no comorbidities.

The following images show the effects of progressive increases of CPAP pressure from zero to 15 centimeters water, the upper airway progressively enlarges with increasing pressure of CPAP (Figure 26). CPAP is like a pneumatic splint for the airway preventing collapse during sleep. It eliminates apneas and hypopneas, decreases arousals, and normalizes oxygen saturation.

In a study by Ueno among 70 OSA patients, CPAP has been shown to improve AHI from a mean of 51.9 to 4.2.

Optimal CPAP pressure, is defined as the level that will abolish apneas and hypopneas, snoring and desaturation in all positions and during REM sleep. It is variable and patient dependent. For our patient, with an initial AHI of 59.8, respiratory events were abolished at CPAP level of 17 centimeters water to an AHI level of 2.8.

In another study by Antic on moderate to severe OSA patients on CPAP for 3 months. 70% exhibited normal sleep latency, and 60% had normal ESS scores, and 35% had normal functional outcomes of sleep questionnaire or FOSQ.

In a study at our institution, CPAP treatment in moderate to severe OSA patients resulted in significant improvements in ESS and FOSQ scores.

In our patient, CPAP for eleven months achieved the following, a decrease in FOSQ score from 80 to 87 and normalization of ESS from 19 to 9.

Other than improvements of symptoms of disrupted sleep, CPAP provides benefits on the patient’s comorbidities. In the study by Sharma and associates, 86 metabolic syndrome patients on CPAP for 3 months showed statistically significant reductions in FBS, triglycerides, hemoglobin A1C, and HDL. On follow-up, our patient showed the following.

Should compliance to CPAP therapy be inadequate, alternative therapy can be instituted. It involves behavioral strategies, oral appliances and surgery. Behavioral strategies include – weight loss, exercise, and sleep positional therapy. Oral appliances such as the Mandibular Repositioning Splint -- to protrude the mandible forward and hold the tongue more anteriorly. And surgery. Surgery is indicated if PAP is inadequate or if the patient exhibits intolerance and when obstructive anatomy compromise other therapies. The common surgical procedures are the following, with uvulopharyngoplasty being the most commonly performed.

The management of obesity primarily focuses on weight reduction.

In a study by Peppard on moderate weight change in patients with sleep disordered breathing, a 10% weight loss causes a 26% decrease in AHI with an increase of 32% if the patient had 10% weight gain.
Weight loss can be approached non-surgically and surgically. Nonsurgical therapy consists of behavioral therapy, low caloric diet, medications and promotion of physical activity. While surgical management centers on bariatric surgery. In this case, can we recommend bariatric surgery for our patient? The answer could be answered by....

A study published by Dixon, where bariatric surgery compared with conventional weight loss therapy showed no statistically significant greater reduction in AHI. Hence, we will not recommend bariatric surgery.

With the management we have given, what lies ahead for PD?

Long term outcomes for OSA patients were studied by Young and associates. In a study on sleep disordered breathing and mortality with an eighteen year follow-up (Figure 27). All-cause mortality adjusted for age, sex, and BMI was significantly increased with SDB severity. The findings also showed a significant high mortality in untreated SDB.

In another study published by Marti on the effects of CPAP on moderate to severe OSA, the data showed a reduction of 40% in all-cause mortality in the treated group against those who did not receive treatment (Figure 28).

A search on the prognosis of his other comorbidities, A BMI of 40-45 reduces life span by 8-10 years. And for untreated early-onset severe kyphoscoliosis, there is significantly increased mortality from respiratory failure or cardiovascular diseases compared to the general population with increased risk of death after 40 years of age.

Presented was a ménage a trois of obesity, kyphoscoliosis and obstructive sleep apnea. Add to the mix the other comorbidities – hypertension, diabetes and dyslipidemia. An unfortunate mix, but a challenge. There are no published data on the prognosis of patients like ours, All his illnesses will eventually take their toll. But we will not be deterred, we will continue... patient education, weight reduction, proper nutrition, control of blood pressure, hyperglycemia, and dyslipidemia and continued adherence to CPAP therapy. Which for now, has had significant effects on the well-being of the patient.

**VIDEO OUTRO by Dr. Ethel Cabrera (voice over)**

In medicine, we live by a rule of William of Ockham, “pluritas non es ponenda sin necessitas” which translates as “plurality should not be posited without necessity.” That means that most of the time, a variety of symptoms can be explained by a single diagnosis.

But we have come across with Hickam's dictum, by Dr. John Hickam, a 20th-century American physician born in the Philippines that simply states -- "Patients can have as many diseases as they damn well please.” At no stage should a particular diagnosis be excluded solely because it doesn't appear to fit the principle of Occam's razor.

Tonight, my colleagues, we have unfolded a ménage a trois of kyphoscoliosis, obesity, and obstructive sleep apnea. In time, the fateful mix will carry a much worse prognosis than any of the three entities alone. We can only hope that our interventions for this young man will carry him through. Thank you and good night.
OPEN FORUM (Q&A)

DR. ABARA: Job well done for our fellows. The floor is now open for your questions and clarifications regarding the case. Dra. Limpin will give the first question.

DR. LIMPIN: I think I did not hear if you did a repeat blood gas of this patient after CPAP therapy. So, can you please give us any data on whether you were able to do that or if none, then of course you cannot give it to us.

DR. FLORES: Thank for you for that question, ma’am. We did a repeat blood gas of the patient after CPAP therapy of about 11 months and the ABG was normal acid base with mild hypoxemia. His pO₂ was 66.3, pH and HCO₃ were normal. The exact values were pH of 7.401, pCO₂ 44.5, pO₂ of 63.6, HCO₃ of 27.0, Base excess of 2.2, and oxygen saturation of 91.2%.

DR. ABARA: Any other questions? Dra. Banzon?

DR. BANZON: One of the latest treatments for obstructive sleep apnea that's recommended is the Servo Ventilation. I would like to know if there is a role for Servo Ventilation for this patient.

DR. CABRERA: Auto-servo ventilation or ASV is indicated for patients with mild to moderate heart failure, obstructive sleep apnea, and central sleep apnea. Unfortunately, this is not recommended in our patient because the patient has no failure symptoms or is not in failure and the patient also does not have any Central sleep apnea.

DR. ABARA: Any more questions or clarifications? The fellows from other institutions can ask their questions? There are microphones in the middle of the hall. Dr. Lucas Uy?

DR. UY: My question is... If you are contemplating surgery in this case, your recommendation was to do uvulopharyngoplasty and bariatric surgery. If you are contemplating doing surgery in this case which one will it be first?

DR. ABARA: Which would you recommend first?

DR. FLORES: The indications for surgery in patients with OSA are the following: if PAP is inadequate and if the patient is intolerant to PAP. With the patient’s good compliance to PAP and with significant results 11 months after, I don’t think we need surgery for the upper airway. As far as bariatric surgery is concerned, it depends on your goals. If your goal is to decrease weight, yes, it may be recommended because for OSA patients, a BMI of 40 is an indication. However with the presence of comorbidities of the patient, I don’t think a lot of surgeons would be brave enough to do so. But if your goal is to improve the AHI which has already been achieved by CPAP alone, we will not recommend bariatric surgery.

DR. ABARA: You still have anything to add Dr. Cabrera?

DR. CABRERA: If we are to choose between uvulopharyngoplasty and bariatric surgery, according to American Academy of Sleep Medicine Guidelines, uvulopharyngoplasty is one of the alternative therapies while bariatric surgery is only used as an adjunctive therapy in patients with OSA.
REACTORS

DR. ABARA: We are lucky to have with us this evening four equally good reactors or experts in their own field. I’ll go with the youngest first. Our first reactor finished her doctor of medicine at the University of Sto. Tomas. She pursued her residency in Internal Medicine at the Veterans Memorial Medical Center and is currently an Adult Pulmonary Fellow in training of the same institution. May I call on Dr. Marie Frances Therese Magnaye-Malicse for her thoughts on the case.

Dr. Marie Frances Therese Magnaye-Malicse: Good evening and thank you for the introduction. I would like to ask for apology for the croaky voice. Anyway, after the case presentation, there are a lot of things that I have to be thankful of... number one, coming from an institution wherein a large part of our population belongs to the geriatric bracket, I am thankful for you that you share this case that of a young patient. The case was well discussed down to the details from the history, the PE, the course as well as all the supporting journals. Now the topic is also relevant since there is increasing incidence of obesity nowadays.

After training when faced in reality we might encounter patients who are obese, those who are a bit deformed speaking of the spine deformity and also those who have excessive daytime sleepiness, even those who do not comply with doctors’ management or recommendation, this case gave us an idea on how to approach such patients. The case presented to us somehow worked up for excessive daytime sleepiness.

We all know separately that obesity, kyphoscoliosis and even obstructive sleep apnea have a negative impact on the pulmonary function. But this conference showed us that these three entities can even exist in just one patient and that makes the course of that patient even more complicated. Not only for the patient but as well to the attending physicians. Lastly, one thing also that I have to be grateful of is for this conference is, because somehow this conference have kept us abreast with the diagnostics in this subspecialty such as the sleep study. With these, thank you and a pleasant evening.

DR. ABARA: Thank you Dr. Malicse. Our next reactor is a Fellow and Past President of the Philippine College of Chest Physicians. She is also a fellow of the Philippine College of Physicians. She finished her Doctor of Medicine in the University of Sto. Tomas and later pursued her residency training in Internal Medicine at Mary Johnson Hospital. Furthermore she pursued her training in Pulmonology and Critical Care Medicine at the Philippine Heart Center and is a medical specialist for the Department of Training and Research of the Philippine Heart Center. She’s already here. Dr. Maria Encarnita Blanco–Limpin.
Dr. Maria Encarnita Blanco-Limpin: Thank you, Issa. Mabilis pumunta yung disabled. Anyway, since I was the second one to be called, then, Issa actually told you a while ago that the first to be called is the youngest and therefore the second will be second to the youngest. So, thank you for the compliment. Anyway, I'd like to first congratulate the presenters for tonight's discussion. I think you did very well and I hope that the fellows in training present here will be able to learn more from how a case is to be presented.

One thing that is very good here is that the fellows were able to not just give you all the literature, everything that you need, the knowledge that you need, but they kept within the case that is being presented.

Whenever we present a case, we must realize that we are not just presenting a clinical case scenario. We are actually presenting a live patient and therefore it is really important that we always keep within the context of the patient that we have right now. So, I think for that we must really congratulate the fellows for really explaining everything giving us the pathophysiology and I'd like to cover certain aspect of this case.

Number one, the presence of kyphoscoliosis presents already some problems with sleep that's why you see you have a patient who has a chest wall abnormality and will therefore have problems of hypoventilation. Now, in the five patients that were discussed by Guilleminault published in 1981 in the CHEST journal, most of these patients actually are not very similar with our patient because majority, as the fellows have already told you that majority of these patients were actually older than our patient who is very young. Although the spinal deformity of these patients is quiet similar. Because as you said that the Cobb angle of this patient is about a hundred. In Guilleminault's five patients, the spinal deformity had a range of 90-115 with an average of 105. So, basically for the information of our spinal surgeon, it is actually within the range. As also seen in patients by Guilleminault, majority of the problems associated with, is that, why is it found in REM sleep? It was already elucidated by Dr. Cuyacot that whenever the patient goes to sleep, normally the main function is carried over by the diaphragm and during sleep particularly in REM, you have actually a decrease in the activity of the respiratory muscles. Now, in patients with kyphoscoliosis, particularly in severe kyphoscoliosis, the diaphragm which is supposed to be the major muscle of respiration, is already at a considerable mechanical disadvantage. And therefore in these patients, they will be dependent more not on the diaphragm but now on the accessory muscles of respiration. However during REM sleep, the activity of the accessory muscles of respiration is also decreased. That explains why in this case you have now hypoventilation that cannot be usually abolished once the patient wakes up. That is the reason why we see in this case the persistence of hypercapnia and hypoxemia even if the patient is already awake.

In patients like this, the most important thing that we need to look at especially if we want to know if the treatment that we are giving is appropriate and at the right dose is to actually take a look at the blood gasses. That is the reason why I asked earlier on the blood gas after CPAP therapy. Now, it is fortunate that the hypercapnia of this patient improved although the hypoxemia is still persistent. So, that means that probably in time, with continued use of positive
airway pressure, the hypoxemia may also be relieved, or the PAO2 of this patient may go back to normal.

How do we know if the patient is adhering to CPAP therapy or not? It is easy for us to just ask the patient but in most instances even in real practice, the patient will tell you that he is putting (adhering to) on his CPAP therapy even if in reality they do not. What is important right now is for you to probably put that memory card that you can already request from the supplier to be part of the positive pressure therapy. From there, you can take note of the number of hours that the patient is on CPAP. Good adherence will be if the patient uses CPAP for more than 4.5 hours. Because studies have shown that more than 4.5 hours will give you improvement in oxygenation, improvement in hypercapnia, and as well as improvements of apnea events.

So, with that thank you!

DR. ABARA: Thank you Dra. Limpin. Our next reactor finished his Doctor of Medicine at FEU-NRMF Institute of Medicine. He has his residency training in Orthopedic Surgery at Philippine Orthopedic Center. Furthermore, he pursued his Spine Clinical Fellowship at Meijo Hospital in Nagoya, Japan. He’s an active consultant of the Spine Surgery Unit of the Philippine Orthopedic Center. He’s a fellow of the Philippine Spine Society as well as a Fellow of Philippine Orthopedic Association. May I call on Dr. Ferdinand R. Bernal.

Dr. Ferdinand Bernal: Thank you very much for inviting me here. I would also like to congratulate the presentors for a very nice discussion. Just to give you a background about scoliosis. Here is an x-ray of the spine (Figure 29) but it’s not clear because of the patient is too obese. Commonly, scoliosis has three curvatures on the coronal plane – proximal thoracic, the main thoracic and the compensatory lumbar curve. I measured the curvature of this patient. The proximal thoracic measures around 65 degrees. The main thoracic is around 115 degrees and the compensatory lumbar curve is around 40 degrees. There is also a severe kyphosis on the sagittal view but however it’s too difficult to see because the patient is too obese.

Just an addition, scoliosis is a three-dimensional deformity besides the kyphosis and the scoliosis, there is also an axial component. This is the one that causes the rib hump. As the thoracic vertebra twists or rotates the corresponding ribs on that side becomes more prominent that is what we see on doing the forward bending test. For this case he has severe rib hump on the right side aside from that we can also note a right sided decompensation on the feature. For surgery, I will describe to you what we are going to do if we are going to do surgery for this case. Unfortunately we were not able to obtain a bending film so we can more or less predict the possible correction or we can assess the rigidity of the curve. For example, this case is a rigid curve, we might do an anterior release and followed by a posterior release. We can do this in one seating or a staged procedure. An additional information for those young patients with scoliosis who just developing the curve, there is a coupled mechanism. As the spine… parang pag nagcucurve siya, parang may coupled twisting. For young scoliotic patients, those curves still has a coupled mechanism but as you age it becomes structural. Even though you correct
the curve the twisted component is hard to correct because the structural changes already occurred

Our case is a 29 year old male. Probably most of the structural changes have occurred in this patient. You might ask me what is the benefit of doing surgery. It was mentioned by the presenter in one of the review of literature for those older than 20 years old, the pulmonary function after doing a scoliosis surgery is not that good. Yes I agree because it will all depend on the amount of correction that we can achieve for this case. If this case is a young patient, we have done as severe as this one in our department. We were able to have a good correction but with for case that is already a 29 year old male, with structural changes. I still don't know how much correction we can achieve. We still need to do some work up to predict the possible correction. Right now if we are going to weigh the benefits and the risks, probably, it will be too risky for our patient.

Regarding the interconnection of obesity, scoliosis and OSA, our case, he was diagnosed scoliotic since 8 years old. So probably it’s a juvenile type of idiopathic scoliosis. For me, I think there is no interconnection between the scoliotic deformity, the OSA and the obesity. Probably the OSA and obesity came in a later time. My thoughts for this case is just to manage it medically first. If we are going to do surgery, we still have to prepare the patient physically because doing a scoliosis surgery is really very extensive. For this case probably it will last more than 10 hours of surgery. Thank you.

DR. ABARA: Thank you Dr. Bernal. For our last but not the least reactor. She’s a fellow of the Philippine Society of Endocrinology & Metabolism as well as a fellow of Philippine College of Physicians. She finished her Doctor of Medicine at FEU-NRMF Institute of Medicine and later pursued her residency training in Internal Medicine at the same institution. She had her fellowship in Endocrinology, Diabetes & Metabolism at the University of the Philippines-Philippine General Hospital. Friends and colleagues let us all welcome Dr. Myla Verastigue-Custodio.

Dr. Myla Verastigue-Custodio: Good evening! I would like to congratulate our presenters for a very well prepared presentation. So for tonight I was given the task to discuss metabolic syndrome particularly the management of obesity. This is my goal for tonight to make it simple. Our patient fulfills the criteria of metabolic syndrome (Figure 30). Central to this is obesity. It is not just obesity but visceral adiposity because the adipose tissue is the one that produces cytokines that leads to the development of the other criteria for metabolic syndrome.

In the Philippines, the incidence of Obesity is 4.9% in 2003. So meaning by this time, it’s more than that. It’s not just BMI which should be looked upon but also waist circumference. The association of BMI-Waist circumference is that as you increase the BMI and waist circumference (Figure 31), you also increase the associated risk such as Type 2 Diabetes, Cardiovascular disease, and hypertension. Now, what are we going to do? In our patient, option
number one is not possible (Figure 32) because of the other comorbidities that are already present. What is our goal? So, weight loss of approximately 10% from the baseline and the target date is, 6 months. So, to achieve the goal, you just have to reduce your total caloric intake by 500 to 1,000 kilocalories per day and that will translate to 1 to 2 lbs. weight loss per week equals to 10% weight loss in 6 months. That will be achieved by low caloric diet with reduction in saturated fats. So we need to decrease the saturated fats in order to decrease the triglycerides and address the cholesterol problem of this patient. We also need to increase physical activity and that is a moderate intensity exercise 30-60 minutes per day, 5 to 7 days per week.

So, I always tell my patients that the first line of treatment for obesity and Type 2 Diabetes is free... diet and exercise. Hindi mo kailangang magbayad. Combination of diet, behavioral therapy, and exercise are very important in the treatment of obesity. And if these fail, there is pharmacologic therapy that is available. In the Philippines, sadly, it is only Orlistat. In the United States last year, they already approve Lorcaserin, Phentermine, and Topiramate on the management of obesity.

And since obesity is already considered a disease, we can expect addition to this management. How about surgical management? If your diet, exercise, and pharmacological therapy fail, you can try bariatric surgery which can definitely decrease the weight. So, if I will be asked by Dr. Lucas Uy if I’m going to advise surgery? First I’m going to ask the presenters how is the patient now? Meron na bang 10% weight loss? Naka 6 months na ba sila in treating the obesity of the patient? And did they already added pharmacologic therapy. Pag meron ng tatlo and the weight of the patient is still the same, probably if the only problem is obesity, yes, surgical management can help. But considering the other comorbidities of the patient, baka hindi siya makalabas sa operating room. So, Thank you very much! The next time you eat, think of your weight, think of your health. Thank you!

**DR. CHAD REY CARUNGIN:** Thank you very much! In behalf of PCCP, I would like to congratulate the training committee especially our chair for the section of Adult Pulmonary Medicine, Philippine Heart Center, Dr. Ayuyao, and our emeritus Dra. De Guia. So, I guess everybody is proud of their fellows and their presentation, and I believe Dr. Limpin was not exaggerating when she mentioned that this is how you present a particular case, not only by history but as well as the dynamics and the interaction of the fellows. Again, she was not exaggerating also that when I say tatak Heart Center, you know the history of Heart Center when they do presentations, not only for the quality but also for the substance and of course they have won several times. When I was coming here I was thinking, meron ba tayong Hall of Famer? Kasi you always get the awards every time. Yun nga Hall of Famer, maybe we can do that particular point. Now, yes, we have to present also.

Before I make some announcements, again when I was coming here, and I heard the song, William actually, then I think everybody knows the song. Actually, it made me tearful because I was a nine year old boy when I sang the Heart Center Hymn here as a nine year old
boy. I was in grade 5 when the Heart Center was inaugurated then, by President and First Lady Imelda Marcos. Again, it brought tears into my eyes and seeing the fellows I have worked with and served under the past presidents, I'm really very happy to be here. So, again that goes to show you how old I am now.

Some announcements, this one will interest you. The research travel grant was raised to 200 dollars for those who are interested fellows in training. To present a paper, oral or poster in any international convention, screened, and approved by the PCCP research committee. It will be a maximum of three requests per institution. Before it used to be 150 dollars, we increased it to 200. Before it used to be just two fellows presenting under one institution, now it’s three and of course it has to be coursed through the PCCP research committee. I think it’s a welcome sign and they will try to fund more on the research.

Again an announcement, our chair for the PCCP Midyear convention Dr. Aniceto will actually be angry if I don’t announce the upcoming PCCP Midyear Convention. Everybody knows about this one. The Midyear Pulmonary Ex Factor will be held at the Grand Caprice, Cagayan de Oro from July 26 to 27. And for the fellows, I urge you to come the day before. It will be on July 25 when we have our Interhospital debate. It will be at 6PM at Mulberry Suites. So, I encourage all the fellows to come early, and then of course the convention will end on a Saturday. The next interhospital symposium will be hosted by Perpetual Succour Hospital on August 30 at the GSK, Zeta Building. And I think why Dr. John is here. He brought along his fellow. He is actually will be presenting for the first time and a very high standard to meet coming from the Heart Center.

Now, again another good news for the fellows, 18th Congress of the APSR 2013 will be held from November 11 to 14 at the Conference Center Pacífico, Yokohama, Japan. That’s one good news. It will be nearer. It will be held earlier than last year, December. And the deadline for abstract will be July 15, 2013. Again, this is the best news. The congress registration fee will be waived for the pulmonary fellows in training. So, that’s good news for us. Of course, the plane fare and stay would be more expensive. Bahala na. Sabi nga ni Dr. De Guia being our Congress for the 2010 APSR. She knows about this of course, she’s a governor for that. So, the announcements were made and I'd like to thank again the Heart Center for holding a very fruitful and very educational presentation as always. I'd like to thank the training committee and I'm glad to see graduates from outside the country are here. So, I would like to thank them for coming here. And I’m sure that you’re very happy with them coming here and visiting you. So, again thank you very much, and then we close our interhospital symposium for today. Of course we will mind the food that we eat at the non-communicable disease and will try to digest it as much as possible.

Thank you very much and good evening!

**DR. ABARA:** The Philippine Foundation for Lung Health, Research and Development in cooperation with the Division of Pulmonary and Critical Care Medicine would like to invite you on our upcoming Biennial Symposium on September 18, 19, 20 at the Crowne Plaza. Dinner is served. Bon appetite everyone!
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APPENDIX

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6