

Subcarinal Angle and Volume

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tracheal bifurcation on chest radiograph is
e. The clinical usefulness of this sign has
al. We correlated subcarinal angle with
the purpose was to find out the diagnostic
ngle for detecting LA enlargement.
re enrolled. All subjects had no diseases
g fibrotic lung diseases, atelectasis, lung
ons. LA volumes were calculated at end-
ic echocardiography (Agilent SONOS
8(A1)(A2)/3 π (L). Briefly, A1 and A2
rea acquired from the apical 4- and 2-
length. Based on 2005 ASE committee
as LA volume larger than 58ml. Upright
valuated, mostly within 1 month of the
ed along the inferior borders of the main
plied to test the difference of subcarinal
enlargement. We used linear regression
inal angle and LA volume. Receiver
med to determine the best cut-off point
t. P value below 0.05 was considered as

6° (36.6-112.9°) and LA volumes were
disclosed subcarinal angles were signifi-
d with normal group ($p=0.0191$). Linear
correlation between subcarinal angles
analysis showed no optimal cut-off point
since textbooks indicated that subcarinal
investigated its power in diagnosis of LA
e predictive value, negative predictive
io and negative likelihood value were
espectively.

subcarinal angle on chest radiographic
d LA volume. However, there was no
n detecting LA enlargement. Subcarinal
tic tool.

Intestinal Neostigmine: Report

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e intestinal pseudo-obstruction, espe-
s with conservative treatment, and
prevent ischemia and perforation of
Neostigmine may be used to rapidly
ar-old woman with MRSA sepsis and
des intestinal obstruction. After failed
e medications, Neostigmine 2mg st i
may be considered neostigmine treat-
failed conservative treatment.
on, neostigmine

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The Influence of the Reduction of Endotracheal Tube Diameter on Ventilator Parameters

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Objectives: The obstruction of endotracheal tube (ETT) is not uncommonly occurring in the patients with mechanical ventilator. However, there are no significant changes of ventilator parameters in the period of partial obstruction of the ETT. Clinically, there will be abrupt deteriorating on the respiratory condition of patients till a breakpoint reach. Nowadays, there are no effective methods to detect early clinically relevant changes of the ETT's diameter or flow inside. We design an experiment to test whether any detectable changes of the ventilator parameters, while progressively narrowing the diameter of ETT.

Method and Material: There are 5 different size (6.0 ~ 8.0) of unused ETT for measurement. The distal ETT is connected to an adult test lung and the model ventilated with a Bird 8400 series ventilators in constant settings. The reduction of the outer diameter of ETT is accomplished by clamping it with a vernier. Each size of the ETT will be test five times and the outer diameter decreasing in a sequence of 2 mm. The ventilators parameters are checked by a ventilator test system which connected between the ventilator and proximal end of ETT.

Results: The parameter measurements revealed that there is a breakpoint present if the outer diameter change greater than $54.11 \pm 6.3\%$ ($\Delta d/D\%$). The measured tidal volume (ml), respiratory rate (rate/min), peak flow (L/min) and peak inspiratory pressure (cmH₂O) are 468.23 ± 9.6 ml, 10.17 ± 0.04 , 41.33 ± 0.9 L/min and 37.05 ± 1.1 cmH₂O in all size of ETT respectively.

Discussion: In this experiment, it is concurring the result in other literatures. The obstruction of the ETT cannot be detected before respiratory distress noted on mechanical ventilated patients. Our results showed that subtle deviate of tidal volume from the initial measurement about $1.88 \pm 1.2\%$. Intriguing, the order of diameter change is not related to the size of ETT. The percentage change of diameter is greatest in the ETT 7.0 before breakpoint reach. It may due to the mechanical property of ETT per se but the mechanism still not known. In conclusion, it is worth to further investigate the ventilator parameters and diameter changes, in the used endotracheal tube with sputum impaction.

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SDF-1alpha 在小膠質細胞調控 interleukin-6 之機轉 SDF-1alpha Up-regulates Interleukin-6 Through CXCR4, PI3K/Akt, ERK, and NF-kappaB-dependent Pathway in Microglia

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Stromal cell-derived factor-1 (SDF-1), also known as CXCL12, and its receptor CXC chemokine receptor 4 (CXCR4) express in various kinds of cells in central nervous system. The SDF-1/CXCR4 signaling pathway is regulated by diverse biological effects. SDF-1 is up-regulated in the ischemic penumbra following stroke and has been known to be associated with the homing of bone marrow cells to injury. However, the effect of SDF-1 α /CXCR4 on cytokine production in microglia is mostly unknown.

We demonstrated that SDF-1 α enhanced IL-6 production in both primary cultured microglia and BV-2 microglia. We further investigated the signaling pathway involved in IL-6 production stimulated by SDF-1 α in microglia. SDF-1 α increased IL-6 production in both protein and mRNA levels. These effects were attenuated by ERK, phosphatidylinositol 3-kinase (PI3K), NF- κ B inhibitors, and I κ B protease inhibitor. Stimulation of microglia with SDF-1 α also increased Akt and ERK1/2 phosphorylation. In addition, SDF-1 α treatment also increased I κ B kinase α/β (IKK α/β) phosphorylation. I κ B α phosphorylation, I κ B α degradation, p65 phosphorylation at Ser²⁷⁶, translocation of p65 and p50 from cytosol to nucleus and κ B-luciferase activity. Moreover, SDF-1 α -mediated increase of κ B-luciferase activity was inhibited by pre-transfection of DN-p85, DN-Akt or DN-ERK2. Increase of IKK α/β phosphorylation and binding of p65 and p50 to the NF- κ B element were both antagonized by PI3K and ERK inhibitors.

Our results demonstrate a mechanism linking SDF-1 α and IL-6, and provide additional support for the notion that SDF-1 α plays a regulatory role in microglia activation.

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