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# 袖状胃切除术改善患者肥胖及代谢的研究进展

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## 摘　要

肥胖症的患病率已发展为全球性流行病，成为当今社会不可忽视的慢性疾病。肥胖与多种代谢性疾病的发生密切相关，不仅严重危害身心健康，也对社会带来了显著的经济负担。因此，如何有效治疗肥胖及其相关合并症已成为社会关注的核心问题。袖状胃切除术(sleeve gastrectomy, SG)是一种常见的减重与代谢手术，主要通过减少胃容量、调节胃肠激素分泌、重塑胃肠道微生物群来改善体重和代谢功能。SG经历了数十年的发展，手术技术日渐成熟。尽管该手术操作简便，但其对胃的解剖结构与生理功能的改变，及其在手术效果中的具体作用仍尚不明晰。本文通过收集并分析相关文献，探讨胃容积变化、胃动力变化及胃分泌功能变化在SG术后体重减轻和代谢改善中的作用机制。

## 关键词

袖状胃切除术，胃解剖，胃生理，体重减轻，肥胖相关合并症

# Research Progress on Sleeve Gastrectomy in Improving Obesity and Metabolism in Patients

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## Abstract

**The prevalence of obesity has developed into a global epidemic and become a chronic disease that**

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**cannot be ignored in today's society. Obesity is closely related to the occurrence of multiple metabolic diseases, which not only seriously endangers physical and mental health but also brings a significant economic burden to society. Therefore, how to effectively treat obesity and its related comorbidities has become a core issue of social concern. Sleeve gastrectomy (SG) is a common bariatric and metabolic surgery, which mainly improves body weight and metabolic function by reducing gastric capacity, regulating the secretion of gastrointestinal hormones, and reshaping the gastrointestinal microbiota. SG has undergone decades of development, and the surgical techniques have become increasingly mature. Although this surgery is relatively simple to perform, the changes it causes to the anatomical structure and physiological functions of the stomach, as well as its specific role in the surgical outcomes, remain unclear. This article collects and analyzes relevant literature to explore the mechanisms by which changes in gastric volume, gastric motility, and gastric secretory function contribute to weight loss and metabolic improvement after SG.**

## Keywords

**Sleeve Gastrectomy, Gastric Anatomy, Gastric Physiology, Weight Loss, Obesity-Related Comorbidities**



## 1. 引言

世界卫生组织(World Health Organization, WHO)将肥胖症定义为对健康产生不利影响的异常或过度脂肪积聚。体重指数(Body Mass Index, BMI)是体重(以公斤为单位)除以身高(以米为单位)的平方，用于定义肥胖。欧洲国家以 BMI ≥ 30 kg/m$^2$ 作为肥胖的标准，在中国将 BMI ≥ 28 kg/m$^2$ 定义为肥胖[1]。自 2000 年以来，高收入国家儿童及青少年的平均 BMI 已趋于稳定，并保持在较高水平。相比之下，东亚、南亚及东南亚地区的 BMI 上升速度加快[2]。预计到 2035 年，全球将有超过 20 亿人患肥胖症[3]。肥胖与多种疾病密切相关，如心血管疾病、2 型糖尿病、睡眠呼吸暂停、非酒精性脂肪肝、心理疾病及癌症等，严重影响人们的生活质量。肥胖症的日益流行已成为全球面临的重大挑战，给人类健康和社会经济带来沉重负担[4]。SG 是一种常见的减肥手术，近年来多项国际指南和系统评价已确认其为治疗病态肥胖及其合并症的有效且独立的手术方法，在减轻体重和改善代谢方面展现出显著效果。通过减小胃容量，手术有助于减少食物(或热量)的摄入量。另一方面，残余胃的适应性重塑、胃排空速率的变化及胃分泌功能的调节，最终通过减缓饥饿感、增强饱腹感，调节脂肪代谢，实现血糖控制，从而帮助机体达到并维持健康体重并改善代谢。正是由于该手术简单的操作能引发复杂的生理代谢改变，深入探讨 SG 后胃的解剖与功能重塑机制，有助于深入理解手术效果，优化治疗策略、提升个体化治疗效果，并为未来的减重与代谢干预提供新的科学依据。

## 2. 袖状胃切除术

SG，通常称为“袖状胃”，最早由 Hess 等人于 20 世纪 80 年代末提出，最初被描述为胆胰分流术与十二指肠转位术的一部分。经过数十年的发展，SG 的手术标准不断优化，并引入微创外科技术(腹腔镜与机器人手术)，通过小切口完成手术操作。这些技术进步显著改善了患者的围术期体验，包括减轻术后疼痛、降低并发症发生率、缩短住院时间及加速术后康复。由于其卓越的减重效果、较高的安全性及相对较低的技术难度，SG 在过去十年间已成为全球范围内最广泛应用的减重与代谢手术。

## 3. 胃的变化

### 3.1. 胃的容积变化

SG 术后胃容量的显著减少是体重减轻的关键因素之一，而术前与术后的胃容积变化可能是决定 SG 减重效果的重要因素。正常情况下，空腹时胃容量约为 50 mL，而进食至饱后，胃容量可增至约 1500 mL。肥胖及暴食症患者的胃容量通常更大[5]。不同国家和地区的 SG 手术标准存在差异。根据手术中使用的胃管尺寸及切胃距幽门的距离，SG 可切除最多约 80%的胃组织，剩余胃(包括袖状胃和胃窦)呈香蕉状[6]。

既往临床研究提出了多种测量剩余胃体积的方法，如亚甲蓝应用的体积测量法、术后悬挂切除胃并用生理盐水填充测量其体积[7] [8]、上消化道造影的几何计算法[9]以及多排计算机断层扫描(multidetector computed tomography, MDCT)的三维重建分析[10]-[12]。无论是上消化道造影的几何类比法，还是 MDCT 的体积测量法，研究结果均一致，SG 术后早期，剩余胃容积约为 100 mL，而在术后 6 个月至 1 年，胃容积可扩张至约 200 mL，且随时间推移呈逐渐递增趋势[10] [11]。

术前体重及 BMI 与切除胃体积显著相关[11]-[13]，这表明体重较重的患者通常具有更大的胃体积，且切除更大的胃体积可能对体重减轻具有更重要的作用。SG 术后胃容积的显著减少可能与短期体重减轻呈显著负相关[14]-[16]，约 1/4 的多余体重减轻百分比(percentage of expected weight loss, %EWL)变异性可由剩余胃容积的变异性解释[17]。然而，长期随访观察发现，这种相关性逐渐减弱[15]，可能与术后胃容积的逐步扩张有关。此外，尽管可调节胃束带术及内镜减重治疗(如胃内球囊、内镜下袖状胃成形术等)同样减少胃容量[18]，但其减重效果仍明显低于 SG。这表明，SG 的减重及代谢改善作用不仅仅依赖于胃容积的减少及热量摄入的限制。

### 3.2. 胃的动力变化

人类的胃可根据功能区域划分为近端胃(或称头区)和远端胃(或称尾区)。近端胃包括胃底和不同部分的胃体，主要负责调节胃容量；远端胃包括胃体的其余部分和胃窦，主要参与食物的搅拌和推进；幽门括约肌则调节食糜进入十二指肠[19]。胃的神经支配来自交感神经和迷走神经的副交感神经，主要分布在胃小弯侧。SG 切除胃大弯侧，因而对神经分布的影响较小。胃的运动形式包括：容受性舒张、蠕动和紧张性收缩。容受性舒张是指胃(主要是近端胃)在食物刺激下，反射性地扩张，从而能够接纳大量食物而胃内压力不会显著升高；蠕动主要发生在远端胃，平滑肌的纵向收缩向幽门方向传播；紧张性收缩则是胃壁平滑肌处于一定程度持续的缓慢收缩状态。

手术的限制作用改变了胃组织的机械反应。研究表明，在容量轻微增加的情况下，剩余胃的压力显著升高，同时剩余胃的体积仅略小于术前胃体积的 10%，并未出现为了接纳更多食物而显著增加胃容积的现象[8]。SG 切除了几乎所有的胃底和绝大部分胃体，这可能切除了发生容受性舒张的主要功能区，因此术后进食刺激下，剩余胃的扩张受到显著限制。剩余胃的直径越大，其扩张能力也越强[20]。这表明，SG 术后体重减轻的机制主要归因于热量摄入的受限，这与剩余胃容量小、顺应性降低及腔内高压的协同作用密切相关。同时，剩余胃腔内压力增高，引起胃壁机械刺激，通过胃－脑轴激活不同的胃受体，这可能影响饱腹感[21] [22]。

SG 术后，胃排空速率加速[23]-[28]。剩余胃可分为两个功能不同的部分：一个基本不动的袖状胃和一个加速排空的胃窦[29]。理论上，胃蠕动频率由胃平滑肌的慢波节律控制，胃的慢波起源于胃大弯上部，而 SG 切除胃大弯侧，可能阻止慢波的产生。摄入食物后，袖状胃腔内压力增高，胃壁的紧张性收缩可能成为袖状胃推动食物的主要动力。然而，胃蠕动并非由单一的、解剖学上定义的起搏器控制，而是受卡哈尔氏间质细胞(interstitial cell of Cajal, ICC)影响。在 ICC 中，起搏周期起始于 ANO1 通道的激活，进而激

活自发的瞬时内向电流(即自发的局部 $Ca^{2+}$释放事件)，去极化激活 CaV 3.2 通道，导致慢波的短暂上行去极化阶段。然后，通过 $Ca^{2+}$诱导的 $Ca^{2+}$释放和 ICC 中局部 $Ca^{2+}$瞬时变化的集合维持 ANO1 通道的激活并将膜电位维持在 $Cl^{-}$离子的平衡电位附近，形成持续几秒钟的长时间平台期。当 $Ca^{2+}$储备耗尽、$Ca^{2+}$释放停止、ANO1 通道失活时 ICC 发生复极化，平台期结束。而胃肠道平滑肌细胞缺乏这些通道，ICCs 在肌肉层和肌间神经丛中形成网络，慢波通过 ICCs 网络进行传播，并通过间隙连接传导到平滑肌细胞。平滑肌细胞去极化并激活电压依赖性 $Ca^{2+}$通道(主要是 CaV1.2)，导致 $Ca^{2+}$内流和兴奋－收缩耦合[30]-[32]。胃窦也能产生慢波[32]，ICCs 网络可能解释了胃窦运动不受影响的原因，胃排空加速可能与胃窦的运动有关。生长抑素对胃的运动有很强的抑制作用，生长抑素分泌的减少可能是 SG 术后胃排空加速的原因之一。

正常情况下，食物在胃内经过机械性和化学性消化，水解和研磨形成食糜。SG 后，胃运动发生变化。同时，由于进食时胃酸分泌减少，胃泌素分泌代偿性增加，进一步促进未消化食物的快速排空，从而刺激胰高血糖素样肽-1 (glucagon-like peptide-1, GLP-1)和肽 YY (peptide YY, PYY)等饱腹感相关激素水平升高，更易产生饱腹感。SG 后 1 年，胃排空延缓与体重减轻不良相关[33]。

## 3.3. 胃的分泌功能变化

胃对食物的化学性消化是通过胃黏膜中多种外分泌腺细胞分泌的胃液来实现的。胃黏膜中有三种外分泌腺：贲门腺，为黏液腺，位于胃与食管连接处宽 1~4 cm 的环状区；泌酸腺，为混合腺，存在于胃底的大部及胃体的全部，包括壁细胞、主细胞和颈黏液细胞；幽门腺，分泌碱性黏液，分布于幽门部。另外，胃黏膜内还含有多种内分泌细胞，通过分泌胃肠激素来调节消化道和消化腺的活动。常见的内分泌细胞有：G 细胞，分泌促胃液素和促肾上腺皮质激素样物质，分布于胃窦；δ 细胞，分泌生长抑素，对促胃液素和胃酸的分泌起调节作用，分布于胃底、胃体和胃窦；肠嗜铬样细胞，合成和释放组胺，分布于胃泌酸区内。

### 3.3.1. 消化液的分泌减少

SG 不仅改变了胃的解剖结构，还对其分泌功能产生了深远影响。在高脂饮食大鼠模型中，胃黏膜功能的失活导致体重、内脏及皮下脂肪组织的蓄积减少，同时肝脂肪变性显著降低，并伴随脂质代谢的改善。与假手术大鼠相比，VSG 大鼠表现出类似的代谢变化[34]。理论上，SG 对贲门腺和幽门腺的解剖组织影响较小。术后，食物更易对胃壁产生机械性刺激，并通过迷走－迷走反射及壁间神经丛的代偿性作用，促进 G 细胞分泌促胃泌素[35]。这一过程进一步刺激胃液(包括盐酸、胃蛋白酶原、内因子、黏液及碳酸氢盐)的分泌。然而，由于 SG 导致泌酸腺组织明显减少，胃液的总体分泌量下降，进而显著削弱胃的消化能力。SG 后，胃的酸性环境在一定程度上得以保留，但各阶段的 pH 值均较肥胖患者有所升高[28]。组胺是强效的促胃酸因子，由肠嗜铬样细胞(enterochromaffin-like cell, ECL)分泌，并以旁分泌方式通过 Gs-AC-PKA 信号通路作用于相邻壁细胞的 $H_2$ 受体，诱导壁细胞分泌胃酸[36]。SG 后，剩余胃胃底黏膜的深部结构发生重塑，腺体中壁细胞及 ECLs 的占比较术前增加约 30% [35]。此外，SG 后患者 ECLs 的内分泌因子表达水平升高，组氨酸脱羧酶(Histidine decarboxylase, HDC)和色氨酸氢化酶 1 (Tryptophan hydroxylase 1, TPH1)的共表达增加，二者分别为组胺和血清素生物合成的限速酶[37]。SG 后，促胃泌素、壁细胞及 ECLs 的协调性增加，在一定程度上补偿了泌酸腺的损失，并恢复了剩余胃的酸分泌能力。

### 3.3.2. 胃的其他分泌作用和微生物群

瘦素和生长素释放肽。瘦素最初被认为主要由脂肪细胞分泌，近期研究表明胃也可分泌该激素。SG 后，瘦素的分泌水平下降[23]。瘦素缺乏可加速胃排空速率，增强空肠的转运活动，并缩短小肠的转运时间，而外源性瘦素补充可延缓胃排空[5]。生长素释放肽是一种主要由胃底 X/A 样细胞分泌的食欲刺激性

肠道激素，其浓度在禁食状态下升高，而餐后下降[38]。生长素释放肽被认为是 SG 后体重减轻机制的重要调节因子[39]。SG 后，胃黏膜发生重塑，导致生长素释放肽阳性细胞的密度降低[40]。此外，术后机械门控 Piezo 离子通道 1 (Piezo 1)表达上调，并通过 CaMKKII/CaMKIV-mTOR 通路抑制生长素释放肽的合成[41]，最终使血浆生长素释放肽水平较术前显著下降[42] [43]。

瘦素和生长素释放肽参与食欲调控。食欲调节在控制食物摄入量方面发挥关键作用。下丘脑弓状核是食欲调节的关键神经中枢，包含致食欲神经元和致厌食神经元[44]。致食欲神经元表达刺鼠相关蛋白(Agouti-Related Protein, AgRP)和神经肽 Y (Neuropeptide Y, NPY) (AgRP/NPY)，而致厌食神经元则表达阿片促黑色素原(Pro-Opiomelanocortin, POMC)和可卡因-安他非明转录调节肽(Cocaine and Amphetamine Regulated Transcript Peptide, CART) (POMC/CART) [45]。SG 通过调控 AgRP/NPY 和 POMC/CART 神经元，降低瘦素和生长素释放肽水平，同时升高 PYY 和 GLP-1 水平[46]，从而抑制食欲并增强饱腹感。

GLP-1 是一种胃肠道肽激素，由分布于胃肠道的 L 细胞分泌，在回肠和结肠中最为丰富。GLP-1 的核心功能之一是通过刺激胰腺 β 细胞释放葡萄糖依赖性胰岛素，同时抑制 $\alpha$ 细胞对食物摄入的胰高血糖素分泌反应，从而发挥肠促胰岛素作用[40]。SG 术后，营养物质更快速地输送至远端肠道，从而刺激 GLP-1 和 PYY 的分泌，增强饱腹感并促进胰岛素分泌[47] [48]。动物实验显示，接受垂直袖状胃切除术的肥胖大鼠胃窦黏膜中 GLP-1 阳性细胞密度显著增加，并能分泌具有生物活性的 GLP-1 [40]。然而，胃源性 GLP-1 在葡萄糖稳态调节中的确切作用仍需进一步研究。

肠道微生物群作为消化道的组成部分，在体重调控和代谢改善中亦发挥关键作用。人类微生物组计划(Human Microbiome Project)的研究表明，肠道微生物群构成了一个复杂的生态系统，包含约 $10^{13}$ 至 $10^{14}$ 个细菌细胞，每克结肠内容物约含 400~500 种细菌[49] [50]。近期微生物组研究表明，人类肠道微生物群主要由拟杆菌门(Bacteroidetes)和厚壁菌门(Firmicutes)组成。SG 改变了胃肠道的解剖结构并影响了肠道微生态，术后微生物群的丰富度和多样性显著增加，尤其是厌氧杆菌科(Rikenellaceae)、另枝菌属(*Alistipes*)和副拟杆菌属(*Parabacteroides*) [51]。在高脂饮食诱导的肥胖啮齿动物中，SG 后瘦素水平的变化具有统计学显著性，并与术后肠道微生物群丰富度的增加及特定菌群的临床参数变化密切相关[38]。此外，肠道中厚壁菌门(与减重手术前后体重变化相关)及变形菌门(与炎症反应及葡萄糖稳态调节相关)比例的变化与 BMI 下降显著相关[52]。SG 术后肠道微生物群的丰富度和多样性增加，这一变化在 SG 介导的减重和代谢改善过程中发挥重要作用。

## 4. 合并症改善

肥胖与多种代谢性疾病密切相关，包括 2 型糖尿病(或胰岛素抵抗)、心血管疾病(如高血压和心力衰竭)、非酒精性脂肪性肝病、睡眠呼吸暂停综合征，甚至某些类型的癌症[53]。既往研究表明，SG 在各年龄段肥胖患者中均能显著减轻体重，并有效改善肥胖相关的代谢性疾病[54]-[56]。胃不仅是一个功能复杂的消化和分泌器官，同时容纳了肠道微生物群这一复杂生态网络，在能量和代谢稳态调节中发挥关键作用。胃通过多层面的相互作用，包括消化、分泌、神经信号传递及微生物群代谢调节，广泛参与全身能量平衡和代谢稳态的调控。

## 5. 术后并发症

随着减重代谢手术经验的积累和手术技术的提高，SG 的安全性逐步提升，但仍存在一定比例的术后并发症，包括消化道漏、出血、VTE、脱水、消化道狭窄与梗阻、营养不良、胆石症、吻合口溃疡、倾倒综合征、低血糖、食管裂孔疝与 GERD、呼吸衰竭、横纹肌溶解综合征、胰腺炎等[57]。除了常见的术后并发症之外，还有罕见并发症。Malek 等人报告了 SG 后神经系统疾病并发症，包括感觉异常、下肢深部

腱反射消失、肌肉疼痛以及某些病例的运动和敏感缺陷。所有病例在接受了维生素 B1 缺乏症的神经病变治疗后，症状均有显着改善或消退[58]。

## 6. 结论

SG 不仅限制了胃的容量，还改变了胃壁对机械性刺激的感知能力及运动功能，使患者更容易产生饱腹感，从而减少食物摄入并抑制热量摄入。此外，SG 手术削弱了胃的消化与吸收功能，降低食物的能量利用率，从而制造热量缺口，最终导致体重减轻。与此同时，SG 通过影响多种胃肠道肽的分泌及重塑胃肠道微生物群，以多元且复杂的机制调节脂质代谢和糖代谢，并改善机体慢性炎症状态，从而进一步促进体重减轻及肥胖相关代谢性疾病的改善。

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